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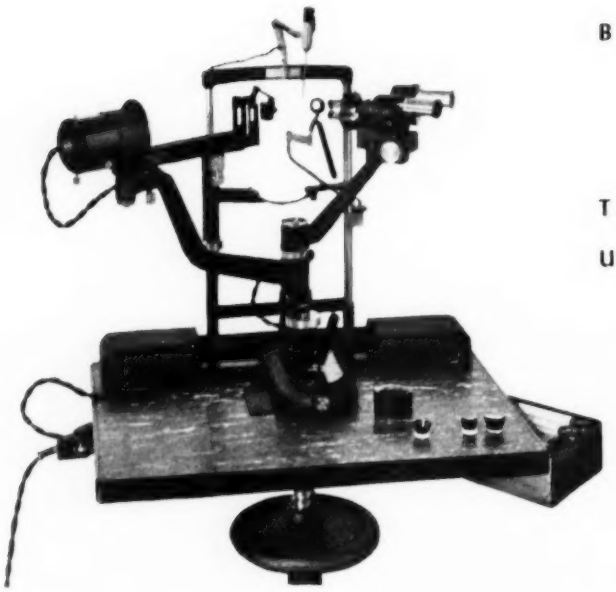

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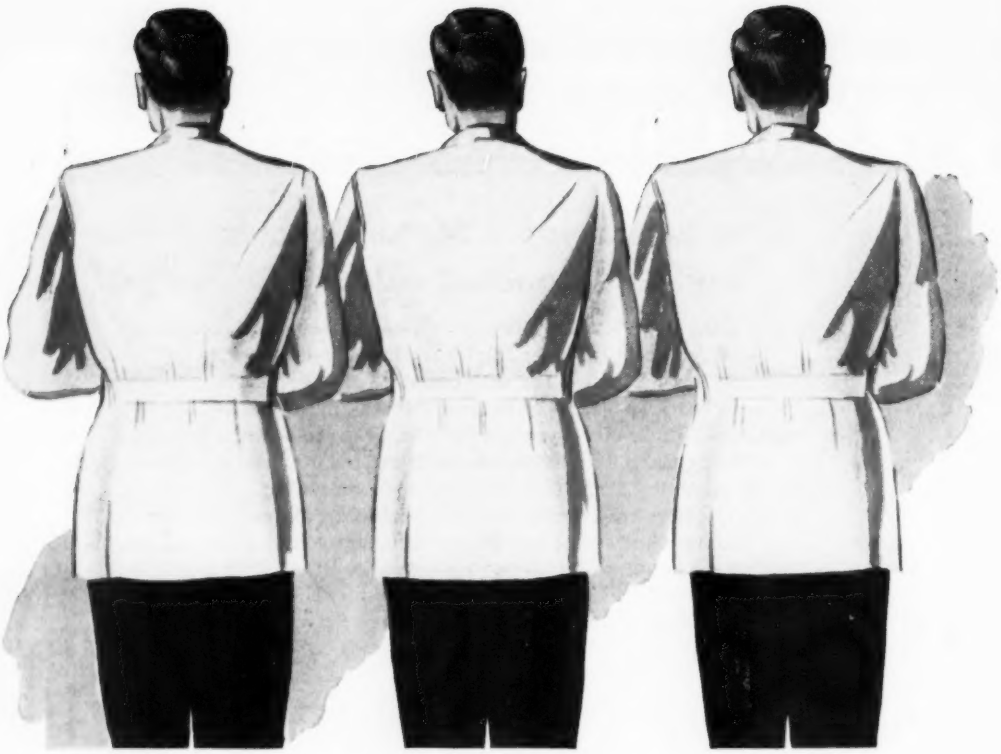

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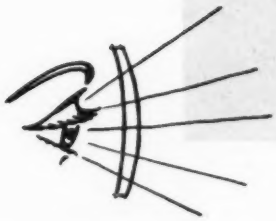

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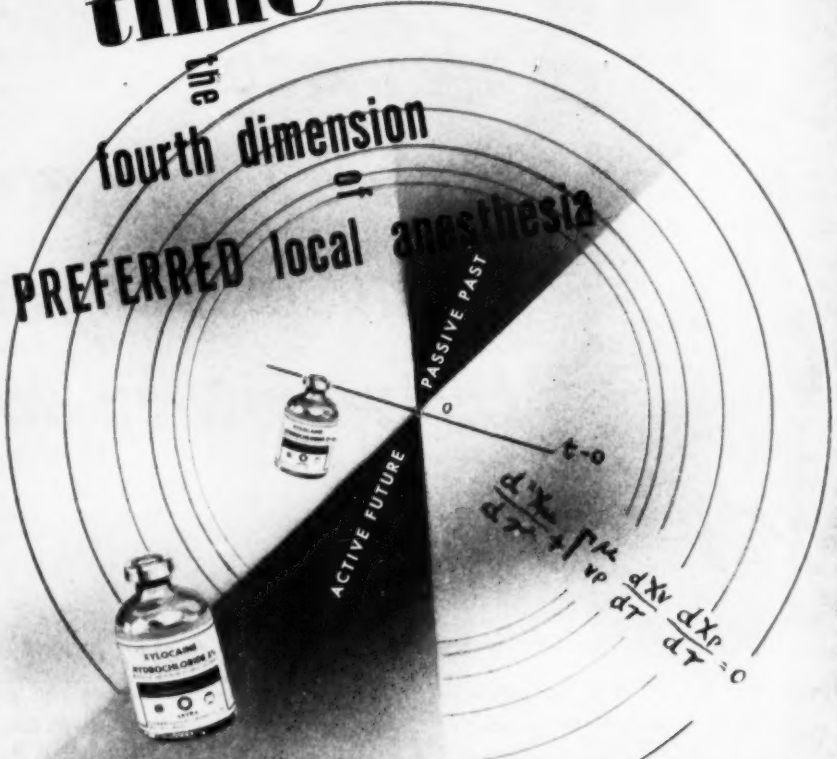

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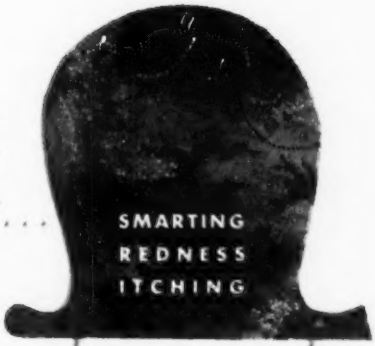

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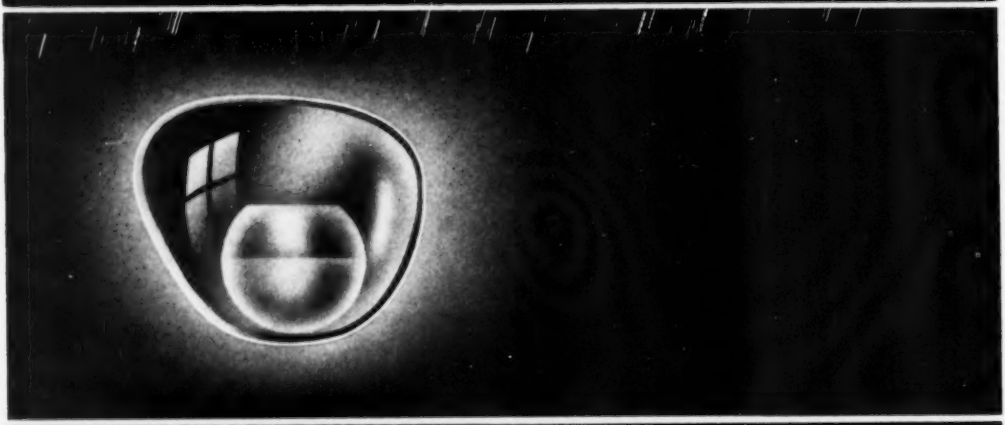

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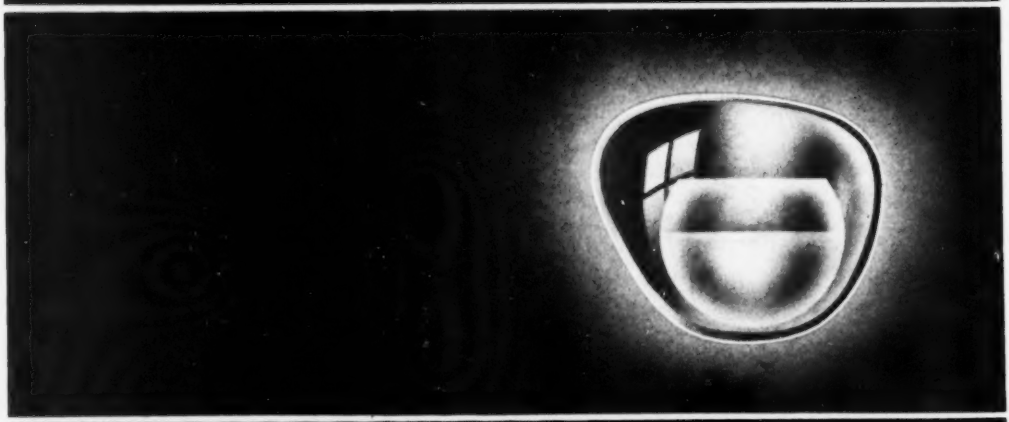

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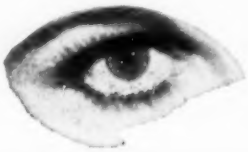

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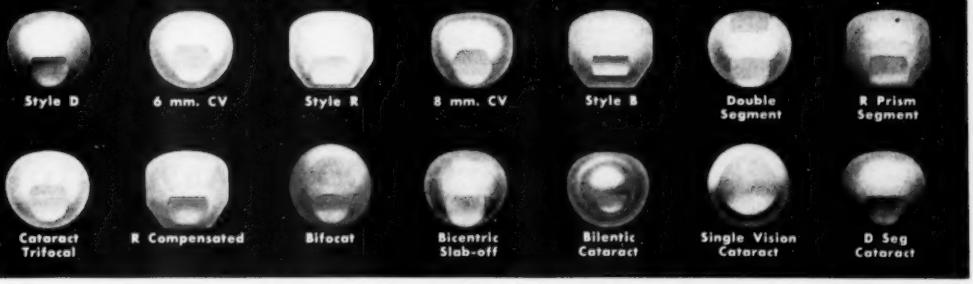

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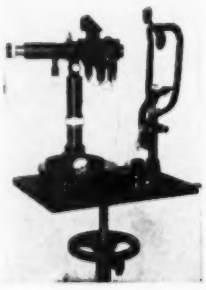

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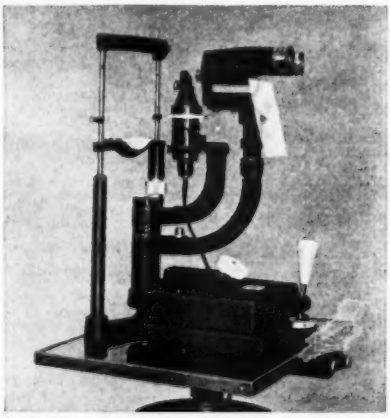

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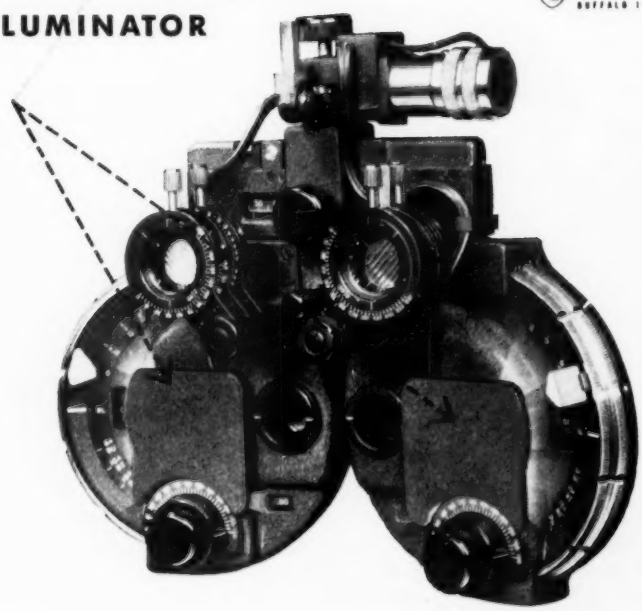

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VOLUME 50 JULY 1953 NUMBER 1

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Editorial

THE BOARD OF ASSOCIATES

WITH THIS issue the three-year term of service of the present Board of Associates comes to a close. On each succeeding month one of the twelve Associates completes his assignment for the Annual Reviews.

The Editorial Board wishes to express its appreciation to each of these men who have made this feature of the ARCHIVES so successful. The task is a laborious and time-consuming one, and it has not seemed fair to impose on one man more than three years' service. Each has made a distinct contribution to ophthalmology by creating a permanent record of the current year's literature in his selected field.

The names of the new members of the Board, who will serve for three years, appear on the front cover.

TRANSPLANTATION OF OCULAR TISSUES TO THE CHORIOALLANTOIC MEMBRANE

Observations on Virus Inoculation

H. P. KIRBER, M.D.
AND
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PHILADELPHIA

THE PRESERVATION of ocular tissues in a living state outside the human body is of interest because such tissues may support the growth of viruses affecting the eye and their mode of action and propagation can be studied. Some viruses have been successfully cultivated in animals or in the developing chick embryo (those of herpes simplex, vaccinia, lymphogranuloma venereum, epidemic keratoconjunctivitis); others, such as the viruses of trachoma and inclusion conjunctivitis, have up to now not been grown with certainty outside the human body.

This study deals with experiments in a large series of mammalian ocular tissues transplanted and kept viable on the chorioallantoic membrane of the developing chick embryo and the infection of such transplants with pathogenic ocular viruses.

MATERIALS AND METHODS

In 1912 Murphy [1] successfully grafted mammalian tumor tissues onto the chick embryo. Since then a number of normal and tumor tissues from different species have been grafted upon the chorioallantoic membrane of the developing chick embryo. Such tissue grafts have been shown to remain viable enough to support the growth of viruses. Goodpasture, Douglas, and Anderson [2] and, later, Blank, Coriell, and Scott [3] reported that intact human skin grafted on the chorioallantoic membrane and infected with the viruses of vaccinia, smallpox, and herpes simplex showed inclusion bodies typical of these viruses. These are viruses which propagate in the human skin and also in the chick embryo. Propagation of viruses which are adapted to the transplanted tissue but not to growth in the chick embryo has

This study was supported by grants from the National Council to Combat Blindness, Inc.

From the Children's Hospital; the Department of Ophthalmology, University of Pennsylvania School of Medicine, and the Department of Bacteriology, Woman's Medical College of Pennsylvania.

1. Murphy, J. B.: Transplantability of Malignant Tumors to the Embryos of a Foreign Species, J. A. M. A. **59**:874-875 (Sept. 14) 1912.

2. Goodpasture, E. W.; Douglas, B., and Anderson, K.: A Study of Human Skin Grafted upon the Chorio-Allantois of Chick Embryos, J. Exper. Med. **68**:891-904 (Dec.) 1938.

3. Blank, H.; Coriell, L. L., and Scott, T. F. M.: Human Skin Grafted upon the Chorioallantois of the Chick Embryo for Virus Cultivation, Proc. Soc. Exper. Biol. & Med. **69**:341-345 (Nov.) 1948.

been demonstrated by TenBroeck [4] in the growth of hog cholera virus in minced hog testis implanted on the chorioallantoic membrane, by Goodpasture and Anderson,[5] by Blank, Coriell, and Scott [3] in the production of typical inclusion bodies in human skin grafted onto the chorioallantoic membrane with the virus of herpes zoster, and by Goodpasture and Anderson [6] in the multiplication of the virus of mare abortion in fetal human membranes grafted onto the chorioallantoic membrane. None of these viruses has as yet been grown in embryonated eggs without susceptible tissue grafts.

The possibility of using the transplantation technique in the study of viruses affecting human ocular tissues presented itself. In 1950 we [7] briefly reported successful transplantation of corneal tissue of rabbit and man onto the chorioallantois and infection of the animal grafts with the viruses of vaccinia and herpes simplex. Later the same year transplantation of ocular tissues from the rabbit by Grignolo [8] and cultivation of rabbit cornea and conjunctiva and their infection with the viruses of vaccinia and herpes simplex by Sezer [9] were independently reported. The results of further experiments with human and animal ocular tissues are summarized below.

BRIEF SUMMARY OF PREVIOUS WORK

Chick Embryos.—Embryonated hen's eggs, preferably between 8 to 10 days of age, were used. The shell over the natural air sac was removed, and a small slit was made in the shell membrane with a hypodermic needle. Through this needle was deposited a drop of sterile saline, which seeped through the opening under the shell membrane, thus separating it from the underlying chorioallantois. The shell membrane was then peeled off easily with forceps. In embryos older than 10 days the chorioallantois was exposed by means of the "false air sac" technique.

Eyes.—Rabbits and guinea pigs were killed and their eyes kept for 1 to 20 hours at 4 C. in phosphate-buffered saline solution of pH 7.2 containing 500 units of penicillin and 0.1 mg. of streptomycin per milliliter.

Human eyes were obtained from stillborn infants, newborn infants who died shortly after birth, and a few unclaimed adult corpses. These eyes were kept for transplantation in the same manner.

Preparation of Tissues.—Sterile technique was used throughout all experiments. The cornea was cut off in full thickness at the limbus with corneal scissors and then spread on a cork disc, with the epithelium facing the cork. When the cornea was used *in toto*, a small limbus-based triangle was cut out to permit the cornea to lie flat on the chorioallantois. Sometimes one-half a cornea was used for virus inoculation, and the other half was used as a control.

4. TenBroeck, C.: Cultivation of the Hog Cholera Virus, J. Exper. Med. **74**:427-432 (Nov.) 1941.

5. Goodpasture, E. W., and Anderson, K.: Infection of Human Skin, Grafted on the Chorio-Allantois of Chick Embryos, with the Virus of Herpes Zoster, Am. J. Path. **20**:447-453 (May) 1944.

6. Goodpasture, E. W., and Anderson, K.: Virus Infection of Human Fetal Membranes Grafted on the Chorio-Allantois of Chick Embryos, Am. J. Path. **18**:563-571 (July) 1942.

7. Kirber, H. P.; Kirber, M. W., and Henle, W.: Transplantation of Cornea to Chorio-Allantoic Membrane, with Observations on Virus Inoculation, Proc. Soc. Exper. Biol. & Med. **73**:481-485 (March) 1950.

8. Grignolo, A.: Culture di tessuto corneale, conjunctivale, sclerale e uveale sulla corio-allantoide di embrione di pollo, Boll. ocul. **29**:617-637 (Oct.) 1950.

9. Sezer, N.: Cultivation of Conjunctival and Corneal Tissue on the Chorioallantoic Membrane, Arch. Ophth. **44**:703-710 (Nov.) 1950.

In most experiments, Descemet's membrane was partially removed by scraping with a scalpel. For virus infection the corneal epithelium was prepared by multiple prick with a 24-gauge hypodermic needle. Virus was rubbed on the epithelium with a cotton swab. The cornea was transferred to the chorioallantois with forceps and a section lifter and placed with the endothelial side facing the membrane, preferably over a large blood vessel.

For transplantation of the conjunctiva, small pieces were removed from the globe or the fornices. To prevent the folding up of the thin and contractile tissue, it was found advantageous to spread it on a glass cover slip and place it on the chorioallantois with the tissue between the glass and the chorioallantois.

For transplantation of iris, small pieces of human and animal iris were excised at the root and cut radially from the pupil toward the root. The pieces were put on the chorioallantois with the epithelial side facing the membrane.

The openings in the egg shells were closed with two layers of Scotch tape, and the eggs were incubated at 37 C.

Vaccinia virus was obtained from a commercial source and passed through several passages on the chorioallantois. Herpes virus (HF strain) was obtained from Dr. T. F. McNair Scott, at the Children's Hospital of Philadelphia. Ten per cent suspensions of infected allantoic membranes stored in ampuls in a dry ice chest provided the stock preparations for the experiments.

At various intervals the transplanted tissues were excised with the attached chorioallantois and placed in 5% acetic acid-Zenker's fixative. Paraffin sections were stained with hematoxylin and eosin.

RESULTS

Cornea.—Of 428 corneal transplantations performed, 84 were of human, 289 of rabbit, 49 of guinea pig, and 6 of chicken corneas. Macroscopic and histological changes are described chronologically.

After 24 hours, the corneal graft was found to be thickened to about twice its original thickness and remained transparent. It was not freely movable on the chorioallantois but could be peeled off with ease, without causing hemorrhage. In histological sections the corneal epithelium appeared normal. The stroma was undisturbed in the anterior two-thirds, whereas the posterior third showed lamellar separation, edema of the interlamellar spaces, and invasion by chick leucocytes. The posterior surface of the stroma was in close apposition with the underlying chorioallantois. The remaining parts of Descemet's membrane were thrown into folds and curled up at the cut edges. The chorioallantois reacted to the transplant by hyperplasia of its mesodermal elements; i. e., the membrane was thicker in the area of the transplant and showed denser cellularity. At this stage the membrane epithelium facing the transplant was still intact in most grafts, appearing as a double row of cuboidal cells with oval nuclei. The transplant was apparently kept viable by "plasmatic circulation."[2]

After 48 hours, the transplanted cornea had increased to about three times its normal thickness and appeared bluish and slightly turbid. The corneal luster gave way to a ground-glass appearance. The transplant could be peeled off the chorioallantois only with difficulty. Microscopically, the epithelium appeared to be intact. The anterior third of the stroma showed unchanged anatomical structure, and the posterior two-thirds showed lamellar separation, with edema and numerous chick white blood cells in the interlamellar spaces. The corneal stroma was in firm apposition with the chorioallantois. The ectodermal layer of the chorioallantois had been resorbed in the area of contact except for islets of "epithelial pearls." Chorioallantoic stroma and corneal stroma were fused in the area of contact. Numerous

vascular buds filled with nucleated chick red blood cells extended from the chorioallantois into the corneal stroma, with the remains of Descemet's membrane seeming to form a barrier against the invading chick vessels.

After 72 hours, the graft was about four times its normal thickness. It was then bluish, opaque, and firmly fused with the chorioallantois. The epithelium and the anterior one-fourth of the stroma appeared unchanged, and the remaining part of the cornea showed lamellar separation and an increased number of new chick blood vessels invading the graft. At this stage the vessels extended to the middle portion of the graft. In some experiments India ink was injected intravenously

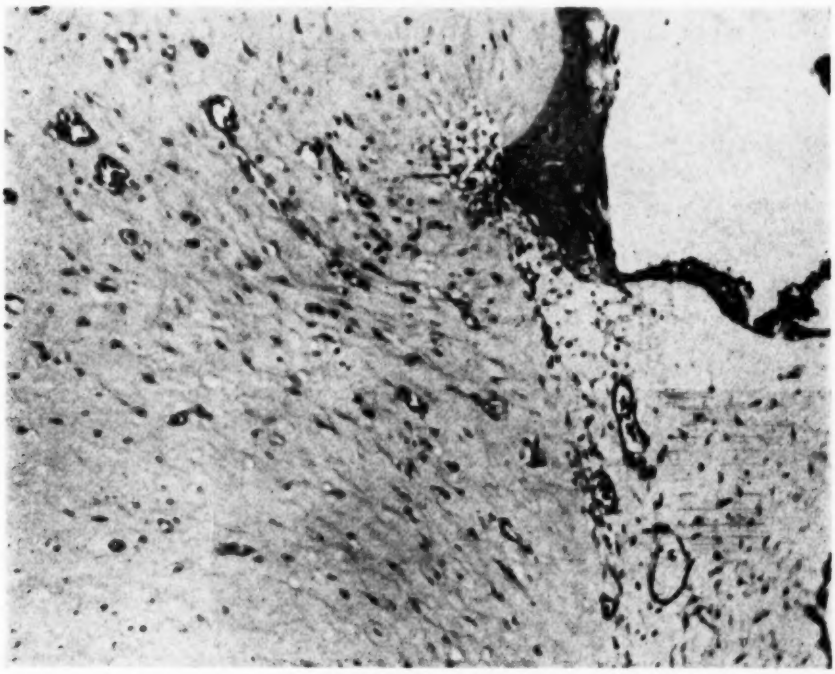

Fig. 1.—Rabbit cornea three days after transplantation onto the chorioallantoic membrane. India ink was injected intravenously into the chick and is seen in the vessels penetrating the graft. The epithelium of the chorioallantoic membrane is resorbed in the area of fusion, and the corneal epithelium is hyperplastic where it is in contact with the chorioallantois; × 150.

into the chick embryo just prior to harvest and could be demonstrated in the vessels of the corneal stroma (Fig. 1). In the area of fusion the mesodermal elements of the chorioallantois and of the cornea were so completely interwoven that they became indistinguishable. The chorioallantois showed marked increase in thickness and denser cellularity. At the margin of the graft the corneal epithelium was completely fused with the chorioallantoic epithelium, and the corneal epithelium was frequently found to be hyperplastic in this area of fusion (Fig. 1). Remnants of Descemet's membrane and epithelial pearls in the area of fusion are shown in Figure 2*A*. Mitosis in the epithelium of a grafted human cornea is shown in Figure 2*B*.

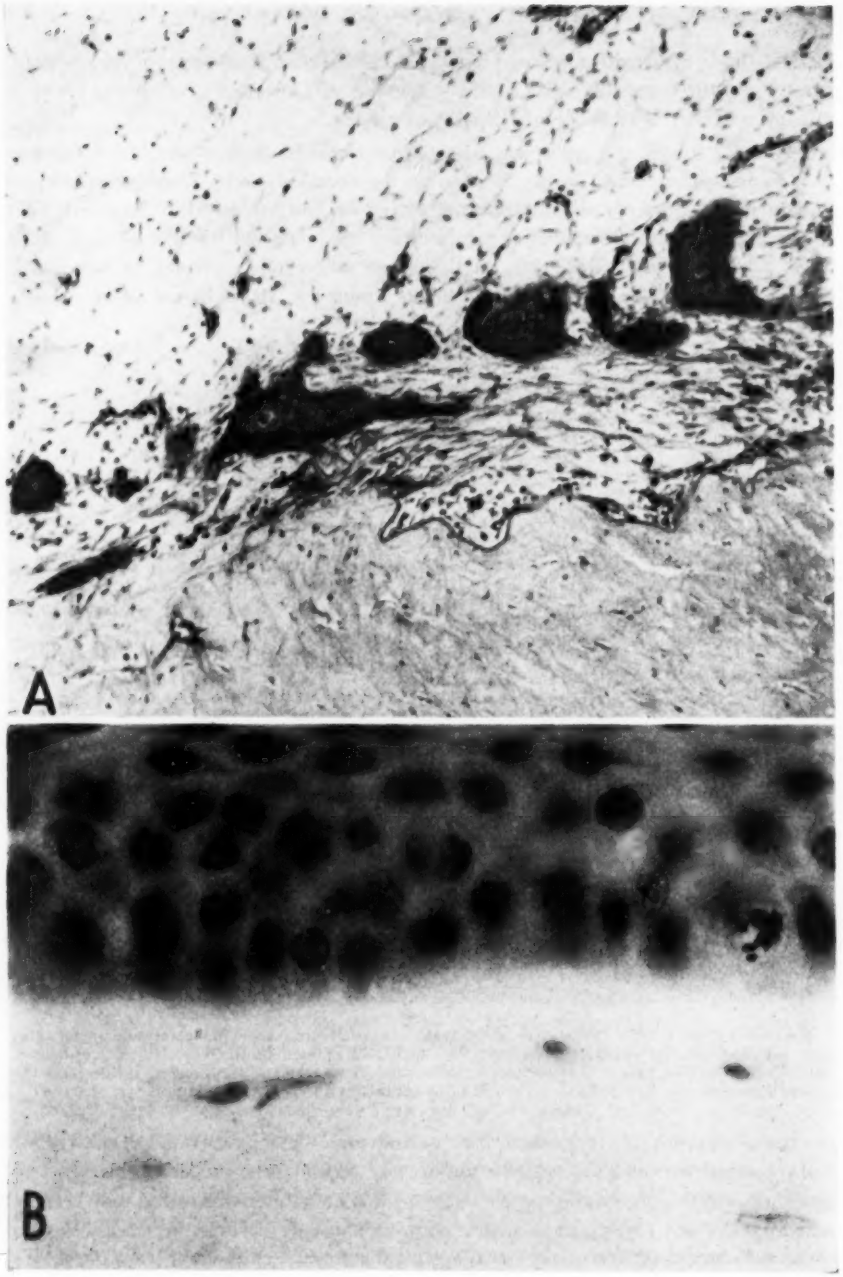

Fig. 2.—*A*, human cornea four days after being grafted onto the chorioallantoic membrane. The epithelial lining of the chorioallantois is resorbed except for islets of epithelial pearls. The fusion of the mesodermal elements is complete except where remnants of Descemet's membrane are present; × 150.

B, human cornea three days after grafting onto the chorioallantois. The epithelium is anatomically undisturbed. Note mitotic figure in the basal cell layer; × 1,000.

From the 4th to the 10th day the graft remained essentially of the same appearance. It was then thick, milky, bluish, and firmly adherent to the chorioallantois. The mesodermal layers of the chorioallantois were completely fused with the corneal stroma. Chick blood vessels were seen to penetrate up to the anterior third of the cornea.

Since the incubation period of the graft is necessarily limited to about 10 days by the hatching of the chick, attempts were made to transfer the graft to younger chick embryos at the end of this period. Fifty-six regrafts of rabbit cornea, 10 of human cornea, 2 of human conjunctiva, and 6 of rabbit iris were made. It has been shown by Blank and associates [8] that human skin can be kept viable for four weeks by such serial regrafts.

The tissue to be regrafted was excised from the chick with the underlying chorioallantois, which was partially scraped off before transfer to a new egg. Such transplants generally showed good fusion, but in the case of the cornea the epithelium was found to desquamate and to separate from the underlying stroma. Grafts in the early stages of fusion (third or fourth day) could be successfully regrafted without loss of epithelium, but in our experience the epithelium could not be preserved for longer periods. Only in one instance was the epithelium of the regraft sufficiently preserved to support the growth of viruses (age of graft, 10 days; age of regraft, 3 days). (In Sezer's experience [9] corneal epithelium could be kept intact in some instances for 20 days by serial passage grafts in early stages of fusion.)

Since maintenance of corneal tissues with intact epithelium for longer periods is essential for the use of this method of cultivation of ocular epithelium viruses, various attempts were made to preserve the epithelium. To maintain adequate moisture, sterile saline was dropped daily on the graft in a number of experiments. Another method of preventing the loss of moisture of the graft was to prepare false air sacs, place the graft on the chorioallantois, and, after allowing time for the graft to become adherent, to add sterile saline to the transplanted tissue. Then the egg was turned, thus placing the graft in direct contact with the shell membrane. Regrafts of tissues maintained in these ways showed no significant difference from tissues grafted by the original method. In several instances the grafts were placed with the epithelium facing the chorioallantois. In such cases complete fusion of the two adjacent epithelial layers was obtained, occasionally with the formation of fluid spaces between them (Fig. 3*A*).

Cortisone has been shown to retard the increase in body weight of chick embryos [10] and to inhibit the vascularization of the cornea.[11] Cortisone acetate in saline suspension injected in 0.2 ml. amounts (5 mg.) into the yolk sac of the chick on the seventh day failed to prolong the hatching period; embryos were underdeveloped but died prior to hatching. Experiments to determine a retarding but less toxic dose are being continued. In a number of experiments cortisone injected in the same manner 24 hours prior to the grafting of cornea onto the chorioallantois on the seventh day showed no retardation of the vascularization of the grafted cornea. In a typical specimen harvested on the ninth day after transplantation, the

10. Sames, G. L., and Leathem, J. H.: Influence of Desoxycorticosterone Acetate and Cortisone Acetate on Body Weight of Chick Embryos, Proc. Soc. Exper. Biol. & Med. **78**:231-232 (Oct.) 1951.

11. Jones, I. S., and Meyer, K.: Inhibition of Vascularization of the Rabbit Cornea by Local Application of Cortisone, Proc. Soc. Exper. Biol. & Med. **74**:102-104 (May) 1950.

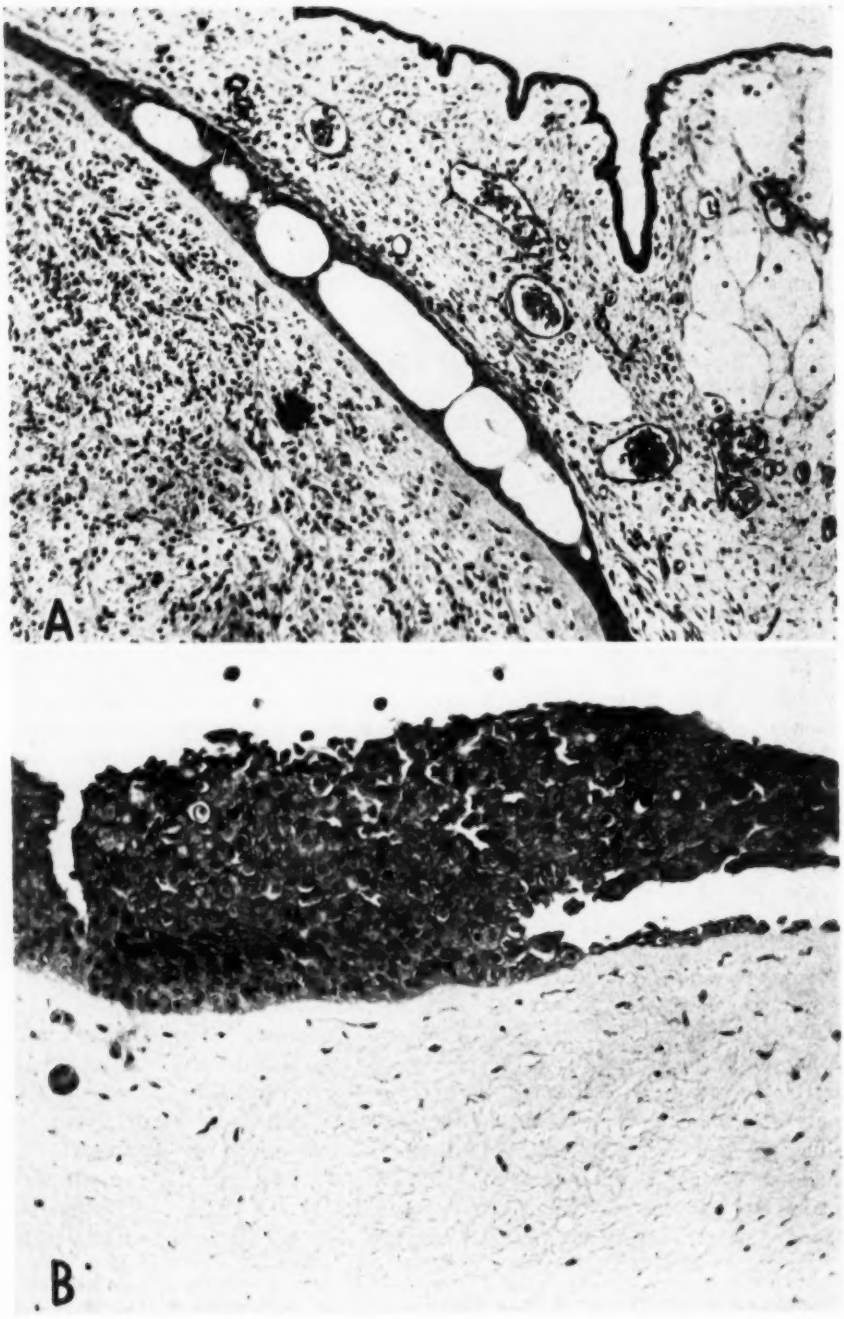

Fig. 3.—*A*, rabbit cornea regrafted from the chorioallantois after three days onto another egg, with the epithelium facing the chorioallantois. It was harvested on the seventh day. Fusion occurs between the two adjacent epithelial layers, and vacuoles form occasionally; × 150.

B, grafted rabbit cornea infected with vaccinia virus by the pinprick method and harvested after 48 hours. There is marked hyperplasia of the epithelium, with formation of numerous cytoplasmic inclusion bodies; × 150.

vascularization had penetrated more than half the corneal stroma, as in the control graft without cortisone. The chief difference between the cortisone-injected and the normal graft was seen in the chorioallantois, which showed edema and thickening, but not the usual cellular hyperplasia, in the area of fusion.

Virus Infection of Grafted Cornea.—Rabbit cornea was infected just prior to grafting with one of the stock viruses by the multiple pinprick method. Infection with the virus of vaccinia resulted in hyperplasia of the epithelium, most pronounced in the polyhedral cell layer (Fig. 3*B*). Typical cytoplasmic eosinophilic inclusion bodies (Guarnieri) could be seen in 24 hours, reaching their height in 48 to 72

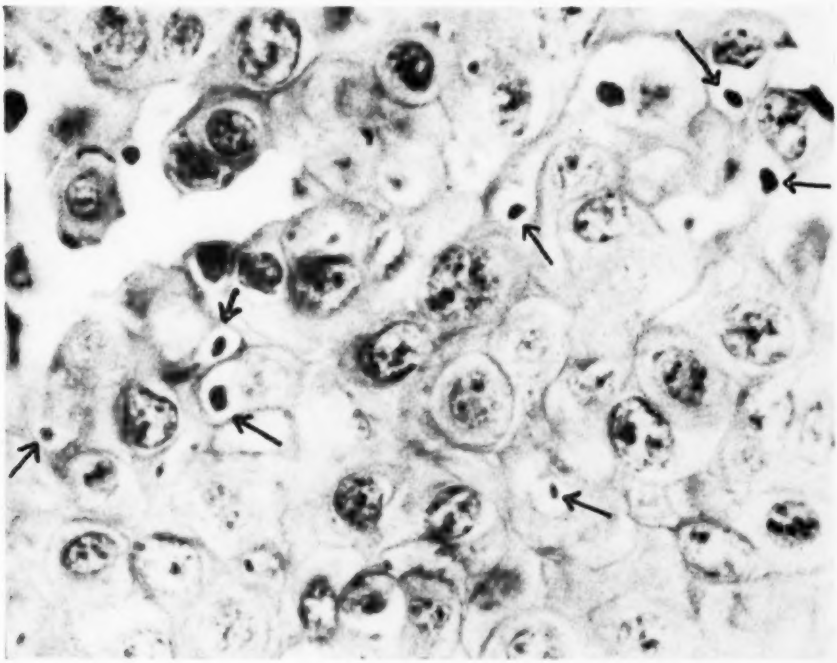

Fig. 4.—Higher magnification of the polyhedral cell layer of the cornea shown in Figure 3*B*. Note characteristic Guarnieri inclusion bodies; × 1,000.

hours (Figs. 3*B* and 4). After infection with the virus of herpes simplex, the epithelium became hyperplastic, and the affected cells showed characteristic eosinophilic intranuclear inclusion bodies (Lipschütz) which reached their height at 72 hours (Fig. 5*A*). The affected cells showed ballooning degeneration with retraction of chromatin around the inclusion body (Fig. 5*B*).

Human cornea was infected with these viruses in a number of experiments. Inclusion bodies could be identified with certainty in the remnants of the epithelial layer only rarely, probably because of the poor viability of the human material at our disposal. A typical Guarnieri inclusion body in an epithelial cell carried into the stroma by pinprick is shown in Figure 6.

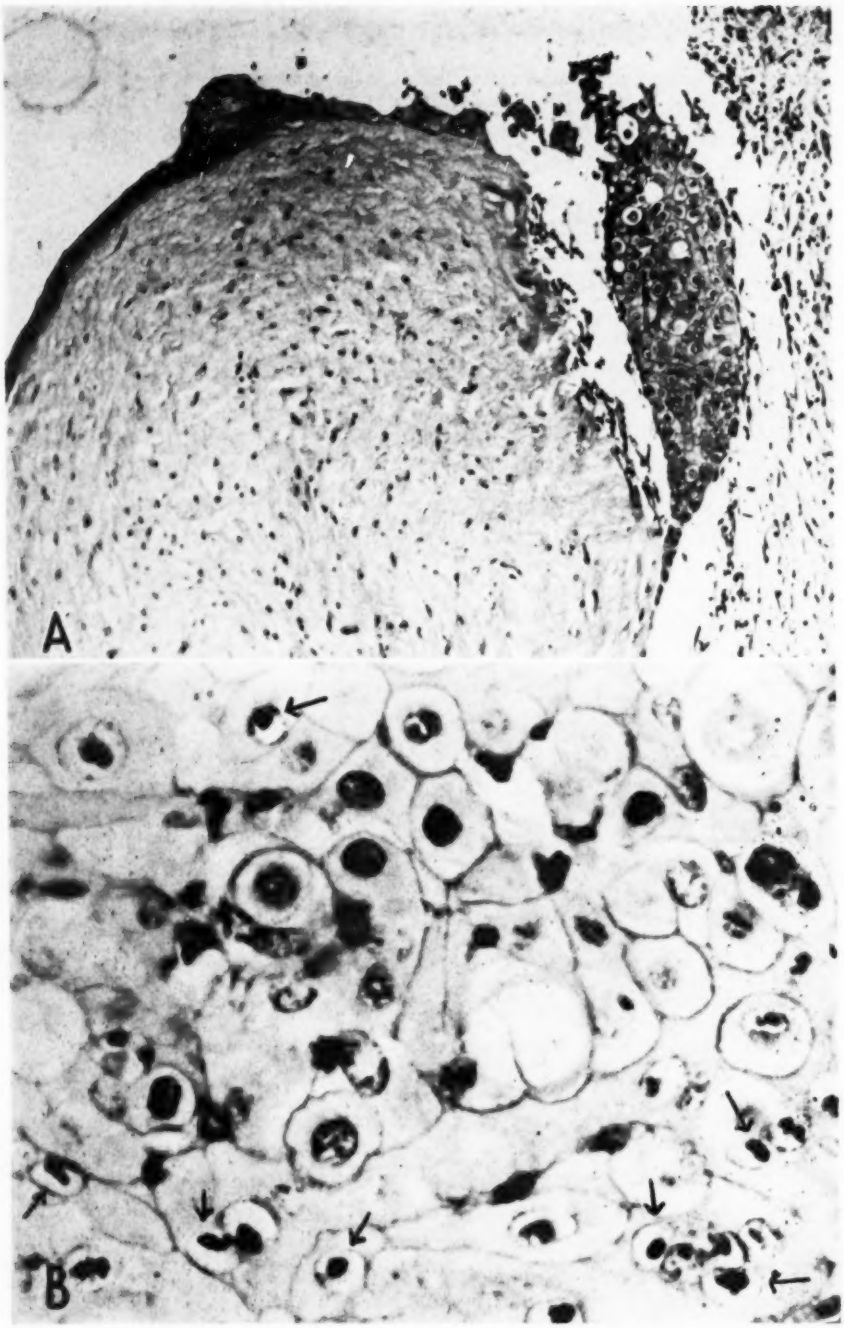

Fig. 5.—*A*, rabbit cornea 72 hours after infection with herpes simplex virus. The epithelium shows marked hyperplasia; × 150.

B, higher magnification of the epithelium shown in *A*. Numerous intranuclear inclusion bodies are seen in the affected cells. The chromatin is concentrated around the inclusion body, and the cells show ballooning degeneration; × 1,000.

Infection of Corneal Grafts with Staphylococcus Aureus.—In order to observe the response of grafted cornea to infection with common pathogens, rabbit cornea prior to grafting was infected by the pinprick method with a virulent strain of Staph. aureus (WP3, 18-hour broth culture). In the cornea of the living rabbit this strain produces a violent inflammation with deep ulcers in 24 hours. When inoculated directly on the chorioallantois, it produces a very strong inflammatory reaction of the chorioallantois, leading to the formation of thick white patches, which on section are shown to consist of a dense accumulation of chick white blood cells. The infection causes the death of the chick in 48 hours. However, attempts to infect corneal grafts resulted in no pathological changes in these grafts, as compared with uninoculated, control grafts of the same age.

Conjunctiva.—Of 208 conjunctival transplantations performed, 76 were of human, 128 of rabbit, and 4 of guinea pig origin. The difficulties encountered in handling this tissue have already been described. The folding up of the tissue could be prevented by spreading it on a glass cover slip. Although such grafts showed good fusion, the epithelial layer was frequently lost when the cover slip was removed. Somewhat better results were obtained by spreading the tissue carefully on the chorioallantois.

The conjunctival graft remained semitransparent for 48 hours. Thereafter it became thickened and turbid. It was adherent to the chorioallantois after 24 hours and firmly fused after 48 hours. Histologically there was complete fusion of the mesodermal elements of the graft and of the chorioallantois, so that it was often very difficult to differentiate chorioallantois from the stroma of the conjunctiva when the tissues had been in apposition for more than 48 hours (Fig. 7). Large vessels filled with nucleated chick red blood cells could be seen in the conjunctival stroma after 72 hours. These vessels may have been preexisting conjunctival vessels which became recanalized (as occurs in the iris transplants), or they may have been newly formed vessels from the chorioallantois penetrating the graft.

The study of the conjunctival epithelium is difficult because small pieces of the grafts, once excised and fixed, are hard to orient for histological sections. In many of the grafts the epithelium was not found on the slides. In some specimens the fusion of the conjunctiva with the chorioallantois led to the formation of one thick membrane, with an epithelium many times the thickness of normal chorioallantoic epithelium, often forming papillae (Fig. 8). Grignolo [8] believed that this epithelium is hyperplastic conjunctival epithelium. However, it shows the characteristics of chorioallantoic more than those of conjunctival epithelium. Hyperplasia of the epithelium lining the inner surface of the chorioallantois was occasionally seen with tissue grafts that sank deeply into the chorioallantois. Such is the case with conjunctival tissue, which often becomes engulfed by the chorioallantois. In such cases the hyperplastic inner lining of the chorioallantois might easily be mistaken for conjunctival epithelium.

Iris.—Of the 75 iris transplantations performed, 24 were of human, 43 of rabbit and 9 of guinea pig origin. In all experiments the transplanted iris became engulfed by the chorioallantois, which was markedly thickened in the area of contact. The chorioallantois showed the usual thickening, with proliferation of its cellular elements and condensation of the stroma. The fusion of these mesodermal tissues was complete. The chick blood vessels were seen between all the layers of

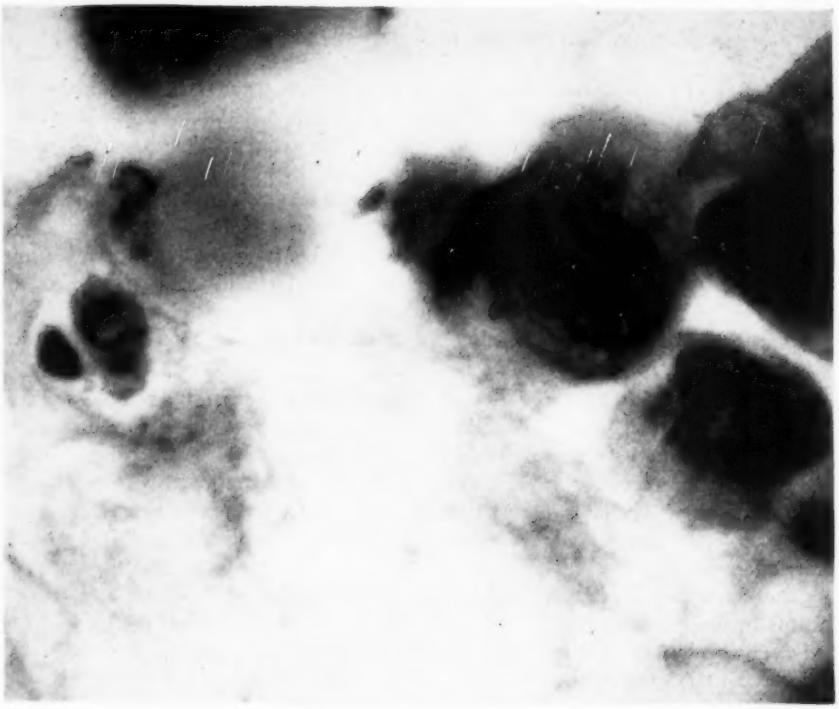

Fig. 6.—Pinprick area in human cornea infected with vaccinia virus and harvested three days after grafting. Cytoplasmic inclusion body (Guarnieri) is seen adjacent to the nucleus; × 2,900.

Fig. 7.—Human conjunctiva seven days after grafting. There is complete fusion of the mesodermal elements, and the conjunctival tissue is traversed by large vessels filled with nucleated chick red blood cells; × 150.

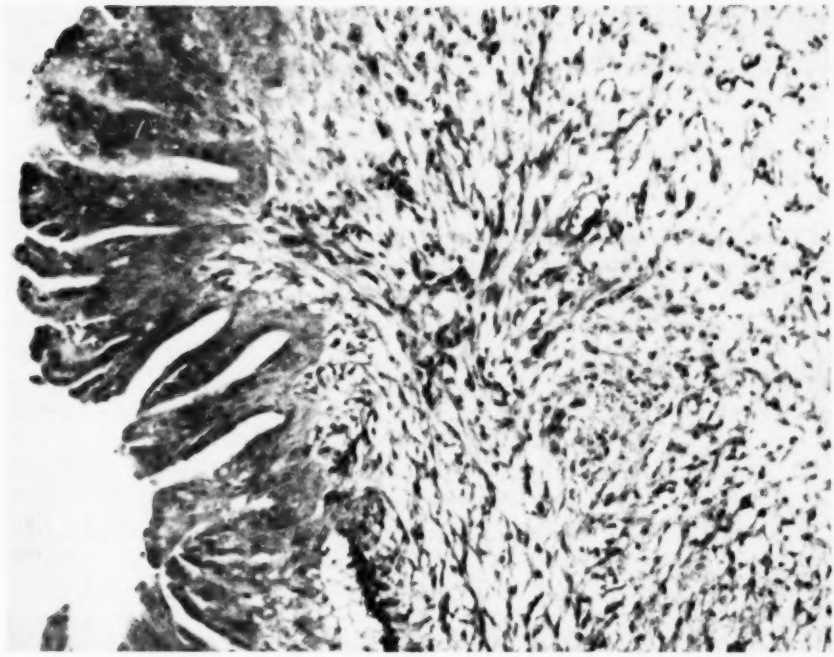

Fig. 8.—Rabbit conjunctiva four days after grafting. The mesodermal elements are fused. The epithelium is hyperplastic and forms papillae; × 150.

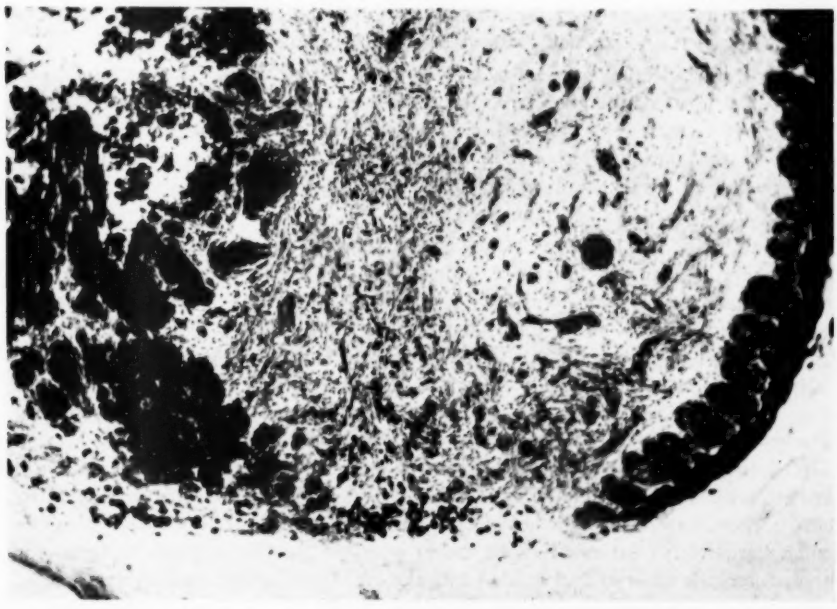

Fig. 9.—Human iris three days after grafting. Blood vessels and fibroblasts from the chorio-allantois have penetrated the graft, and the large iris vessels adjacent to the posterior epithelial layer (dark pigmented line) are filled with nucleated chick red cells; × 150.

the iris, reaching from the posterior epithelium through the stroma to the anterior border layer. The large iris vessels were filled with nucleated chick cells, indicating recanalization. The pigment in the anterior limiting layer of the stroma had become broken up into small clumps (Fig. 9).

COMMENT

It has been shown in this study that animal and human ocular tissues can be grafted onto the chorioallantois of the developing chick embryo and kept viable. The viability of the transplants was shown in several ways:

1. The invasion of the graft by polymorphonuclear leucocytes from the chick and by blood vessels from the chorioallantois could be observed, as well as the appearance of nucleated chick red blood cells in preexisting vessels.

2. The corneal epithelium showed many mitoses and hyperplasia when in contact with chorioallantoic epithelium and when infected with viruses.

3. Epithelial viruses (vaccinia and herpes simplex), which require living cells for multiplication, formed typical inclusion bodies.

4. Control tissues from identical sources, prepared in the same manner as the grafts but placed on a sterile cover slip and thus kept from fusion with the chorioallantois, were dry within 48 hours and on histological section were found to be necrotic.

Several difficulties were encountered with this method. Human ocular material was scarce and rarely obtained before the third day after death. Primary grafts were successful with viable material, but even these freshly obtained tissues were difficult to maintain for several passages. A number of modifications of the technique, in an attempt to keep the epithelium of the regrafts viable, have failed. The influence of cortisone on such grafts is being further studied.

Only a limited number of viruses give rise to the formation of inclusion bodies. Histological proof of the presence of other viruses would depend on the reaction of the infected tissue. It is hoped to study the infection of ocular tissue with such viruses by this means. It should be noted, however, that preliminary attempts to show response in the case of a bacterial agent, Staph. aureus, have so far shown no results. This work is being pursued further.

The ultimate aim of these studies is to transplant viable human ocular tissues into the sterile medium of the chick embryo, which is unable to form antibodies, and to keep these tissues viable through several transplantations. Such tissues could then be infected with viruses from ocular lesions which have so far not yet been grown outside the human body (e. g., the viruses of inclusion blennorrhea and trachoma). Should it be possible to grow these viruses in isolated human ocular tissue on the chick embryo, the advantages would be obvious. Through several passages in human tissues nourished by the chick embryo these viruses may become adapted to propagation in the chick embryo proper. That these viruses have not yet been adapted to a nonhuman host might be due simply to the difficulty in getting enough virus material from a lesion of the human eye during life. Infected human ocular tissues in a series of chick embryos might provide enough material for successful chick embryo and animal inoculation. Once grown in larger amounts, various aspects of these virus infections, thus far unexplored, could be investigated, such as antigenic structure, specific therapy, and prophylaxis. It is also felt that

viruses might be isolated in this manner in the case of other ocular diseases not considered or proved at present to be of viral origin.

It is believed that even at the present stage the method might be of use in areas where fresh human ocular tissues from autopsy material are more readily available.

SUMMARY

Cornea, conjunctiva, and iris from rabbit, guinea pig, and human eyes were grafted onto the chorioallantoic membrane of the developing chick embryo and kept viable. The histological appearance of such grafts is described.

The viruses of vaccinia and herpes simplex gave rise to the formation of characteristic inclusion bodies within 24 to 72 hours.

Attempts to prolong the viability of the grafted tissues are described.

The possibilities and advantages of this method are discussed.

NOTE: Since this report has gone to press, a paper has appeared in which the virus of epidemic keratoconjunctivitis was grown on human cornea grafted onto the chorioallantoic membrane of the embryonated egg.[12]

12. Sezer, F. N.: Cultivation of Virus of Epidemic Keratoconjunctivitis on Chorioallantoic Membrane of Fertile Egg, A. M. A. Ophth. **49**:293-302 (March) 1953.

MONOCULAR APHAKIA

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IN THIS paper I shall endeavor to recall to you as logically as I can certain facts and truisms concerning aphakia which, in my opinion, should be carefully considered by every ophthalmologist in any case before undertaking the management of cataract, especially when the patient is or will be aphakic in one eye only.

A person may be aphakic in one eye while the other eye is (1) normal, with good visual acuity and (*a*) no material refractive error, or with (*b*) notable refractive error and (*c*) good accommodation (young person), or with (*d*) presbyopia; (2) cataractous, or otherwise diseased, with poor visual acuity, with or without correction, and with or without accommodation, or (3) blind, or nearly so.

THE APHAKIC EYE

On removal of its lens, the eye is reduced to the simplest kind of an image-forming instrument. The entire optical system in the aphakic eye consists of the curved corneal surface, separating air from aqueous and vitreous. It represents a high-grade curvature hypermetropia. A change in both the number and the location of the cardinal points and planes of a normal eye occurs when the lens is removed. This change alters the direction of the visual axis, which makes it necessary for the eye to turn about 2 degrees outward to obtain an image at the fovea.

The retinal image, even with the correcting lens close to the eye, is about 30% larger in the aphakic eye than in the normal eye. The size depends on the previous refraction—larger if the eye was myopic than if it was hypermetropic—but always larger than in a normal eye.

The aberrations of the aphakic eye are much greater than those of the lens-containing eye. Normally, the harmful effects of the chromatic and monochromatic aberrations on distinct vision are rendered innocuous by both the physiologic characteristics and the structure of the optical system of the eye. The faults of the corneal surfaces are neutralized to a remarkable extent by the curvature of the surfaces and the nature of the indexes of refraction of the crystalline lens.

The removal of the crystalline lens, especially a senile lens, renders the eye hundreds of times as sensitive to violet and ultraviolet rays as before.

The aphakic eye is static. Total absence of accommodation is very important. There is always some accommodation in a normal eye, regardless of the age of a person, and even the slightest amount is a help in the adaptability of the eye. In a static eye properly corrected for distance, the vision is blurred from 4 meters in. It is only reasonable to suppose that in aphakia the optic reflexes are disturbed.

Read before the Section on Ophthalmology of the College of Physicians of Philadelphia, Oct. 23, 1952.

In addition to the optical and physiological changes naturally produced by removal of the crystalline lens, there are numerous imperfections due to faulty technique or to unavoidable complications, one or more of which might, and usually do, occur after every extraction—for instance, irregular or regular astigmatism, changes in the shape of the pupil or the action of the iris, loss of transparency of the media, or newly formed opacities. Regardless of the dexterity of the surgeon, and even with a satisfactory result of the operation for senile cataract, there remains in nearly every instance a more or less mutilated eye.

The eye becomes a different optical instrument on removal of its lens. It cannot be compared in excellence and adaptability with the normal eye. An entirely new kind of vision is provided, probably as acute as or even more acute than before; but with magnified images, no accommodation, greater aberrations, lessened focal depth, and a changed pupil, which allows a different quantity and quality of light to enter the eye. The patient must accustom himself to the strong aphakic spectacle lens, with its many faults, small field, image distortion, and a newly acquired astigmatism; or to a contact lens and all the bother that goes with it.

The aphakic eye is so different from a normal eye that elderly people seldom, if ever, are able to obtain good single binocular vision with two aphakic eyes when corrected with spectacle lenses. How, then, could an aphakic eye possibly be paired up with a normal eye to make a comfortable, workable team? It cannot be done, whether it is corrected by a spectacle lens, a contact lens, or a minifying lens. Nevertheless, many claims have been made that good binocular single vision, even good stereoscopic vision, from infinity in to 10 in. (25 cm.), in patients of all ages, even to very young persons with good accommodation in the phakic eye, has been given to monocular aphakics by the use of a contact lens; that the retinal image is then of the same size, or nearly the same size, as that in the other, the normal, eye.

As a matter of fact, the retinal image in the aphakic eye, when corrected with a contact lens, is at least 10% larger than that in a previously emmetropic eye, as compared with about 30% when the same eye is corrected with a spectacle lens. This still is not an unimportant size difference. But even if the retinal images were equal in size, good single binocular vision could not be attained for two eyes one of which is normal and the other aphakic. No matter how the aphakic eye is corrected, it can never be made an adequate substitute for a normal, dynamic eye, and mere absence of diplopia does not indicate single binocular vision. While certain devices may apparently, for some unknown reason, enable a patient to obtain single vision, careful examination will show it to be spurious—nothing more than the patient has learned to disregard entirely the image of one or the other eye.

It does not require very careful examination of most case reports to show their unreliability. In many instances the only thing they actually prove is that they were hastily made, and that they are superficial, faulty, and incomplete. Too much is left unexplained.

Consider, for example, the case of a young person with good accommodation and emmetropia in one eye, and with aphakia in the other eye, corrected with a contact lens. The visual acuity is 6/6 in each eye. Suppose he can overcome the aniseikonia and that he can superimpose one image on the other without diplopia. He has, then, a sort of single binocular vision at 6 meters. Even at this distance he will use 1/6 D. of accommodation in the normal eye. As the object is brought closer and closer, the accommodation of the normal eye will be brought more and

more into play and the retinal image will become smaller and smaller but remain distinct, while in the aphakic eye the vision will become increasingly blurred and the image larger and larger. The discrepancy increases until at the reading distance the aphakic eye will require a convex lens of about 2.75 D., while the normal eye will use its accommodation. The aniseikonia will be increased; the prismatic effect of the spectacle reading lens will come into play, and there certainly will not be single binocular vision at the reading distance under these conditions.

All that takes place when one eye is emmetropic is exaggerated if the normal eye is ametropic. Depending on the strength and character of the correcting lens, numerous disturbing factors will be introduced the moment the eye turns from looking exactly along the pole of the spectacle lens. In this case, even if the normal eye is corrected with a contact lens, an extra glass for reading will be necessary for the aphakic eye.

A young person, therefore, with monocular aphakia and with the other eye emmetropic will have to wear a contact lens besides a reading lens in a spectacle for the aphakic eye. The young person with monocular aphakia, and with the other eye ametropic will wear either a contact lens in both eyes with an extra reading lens for the aphakic eye or a contact lens in one eye and spectacles with a single lens for the normal eye and a bifocal lens for the other. Usually in these instances prisms are necessary.

The monocular presbyope, having good vision with or without glasses in the normal eye, is seldom a satisfied person, whatever is done for the aphakic eye, at least not in my experience.

The patient with good visual acuity in his aphakic eye and with vision in the other eye too poor to be of any use is the least bother to himself and to the ophthalmologist.

A one-eyed person is not necessarily unhappy, and not terribly handicapped. Many who have never had binocular single vision go through life and do very well in most occupations and in many sports. One-eyed persons can have a considerable sense of depth and solidity, and judgment of distance. For direct vision one eye is as good as two.

Of course, two eyes with good, comfortable binocular single vision are better than one. Stereopsis is a great advantage, and it would be worth a great deal of trouble to acquire it. In my opinion, however, if a patient already has one useful eye, whether aphakic or normal, it is not worth subjecting him to the hazard, apprehension, nervousness, and cost of an operation, then the fitting of a contact lens, besides a pair of spectacles, and, after all that, a course of training, all in order that he may obtain at best a counterfeit sort of single binocular vision.

PATHOLOGIC PHYSIOLOGY OF STRABISMUS

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STRABISMUS is a disturbance of ocular motility due to a number of separate and unrelated pathologic processes. The thesis I wish to develop is that, although at the present time we do not know what all these processes are, we may obtain a better understanding of strabismus by analyzing where and how each process affects ocular motility.

The neuromuscular mechanism which controls eye movements may be compared to that which moves the limbs. The pathways of each mechanism may be divided into two easily identifiable portions: (1) a supranuclear portion, or upper motor neuron, and (2) a nuclear and infranuclear portion, or lower motor neuron. It makes no difference whether the pathways we are considering are transmitting impulses for what, philosophically, we may choose to call "willed," or voluntary, movements, or whether they transmit reflex, or involuntary, activity, each pathway is divisible into these two main portions, and affections of one portion are always different and usually distinguishable from those of the other.

Figure 1 shows the two portions of the corticospinal pathway to the muscles of the arm. The lower motor neuron begins with cells in the ventral horn of the cord. The neurons from these cells to the muscle are spoken of as the "final common pathway," a term introduced by Sir Charles Sherrington in 1904. The final common pathway is the sole path by which all impulses, no matter whence they come, must travel if they are to act on the muscle fibers. They constitute a public path common to impulses arising from many different sources in the body.

The upper motor neuron is composed of fibers conducting many different influences to the muscles, each fiber constituting a private path from some special source, such as afferent impulses coming into the central nervous system from the various sense organs, motor impulses originating in the Betz cells of the motor area of the cortex, and modifying impulses from reflex centers, such as the vestibular nuclei, all converging on the final common pathway. It is well recognized that lesions of the final common pathway result in restriction of movement of individual muscles, whereas lesions of the upper motor neuron result in complex disturbances usually distributed to groups of muscles serving a common purpose.

The neuromuscular mechanism which controls eye movements is likewise divisible into two portions: (1) supranuclear, and (2) nuclear and infranuclear. Figure 2 shows the pathways for dextroversion, both voluntary and reflex. The supranuclear portion includes all those pathways, afferent and efferent, lying above the level of the

From the Department of Ophthalmology, University of Pennsylvania School of Medicine.
Delivered as the Fifteenth de Schweinitz Lecture at a meeting of the College of Physicians of Philadelphia, Nov. 20, 1952.

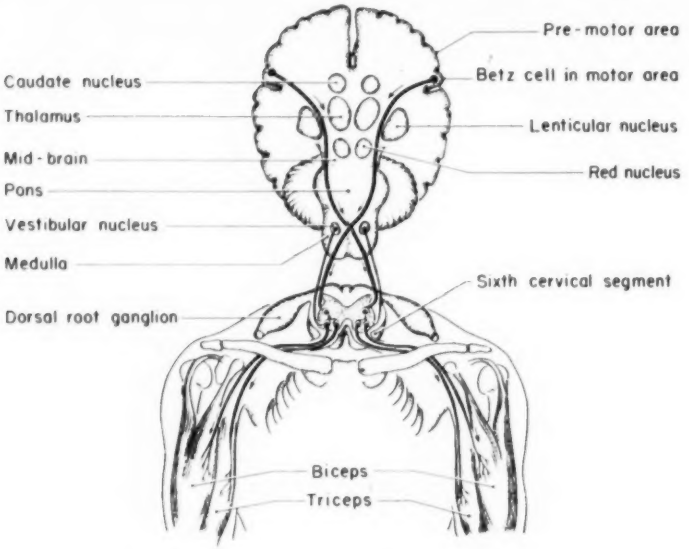


Fig. 1.—Corticospinal pathway to the muscles of the arm.

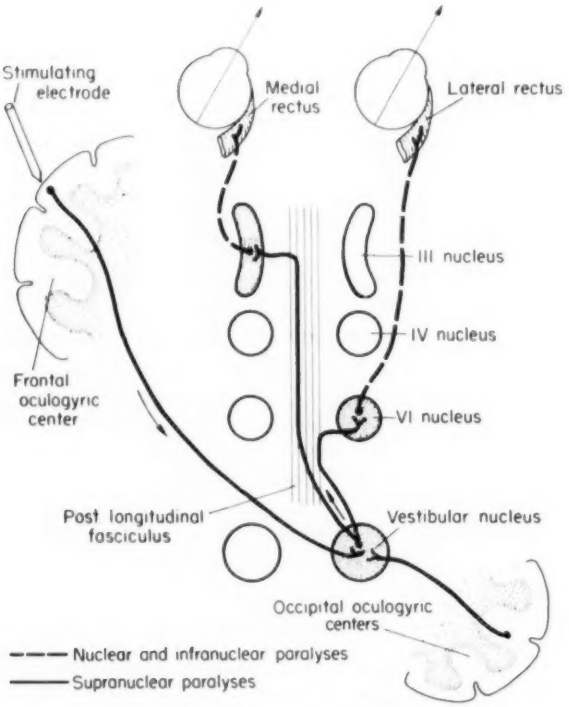


Fig. 2.—Motor pathways for dextroversion.

third, fourth, and sixth nuclei. It includes both voluntary pathways, serving to turn the eyes in any chosen field of gaze, and the various reflex pathways, which keep the visual axes in strict alignment in the various fields of gaze. The nuclear and infranuclear portion includes the nuclei of origin of the oculomotor nerves, the nerves themselves, the neurohumoral mechanism in the synapses and in the motor end-plates, and, finally, the muscles themselves.

Patients with strabismus are commonly divided into two groups: those whose angle of deviation is comitant and those whose angle is incomitant. By comitant strabismus is meant a relatively constant angle of deviation of the visual axes in versions and vergences, regardless of which eye fixates. By incomitant strabismus is meant an angle of deviation which varies in versions or vergences, and with the fixating eye.

An incomitant strabismus is always caused by an insufficient or excessive activity of one or more of the ocular muscles. The abnormal activity of the muscle or muscles may be due to insufficient or excessive innervation, to faulty anatomical relationships, or to the presence of contractures or fibrous bands. From Figure 2 it is apparent that any lesion involving the nuclear and infranuclear portion of the pathway for versions will inevitably result in an incomitant strabismus, since individual muscles will be affected, and therefore the angle of deviation of the visual axes will vary depending upon the field of gaze and upon the fixating eye.

Whenever strabismus is frankly incomitant, the pathologic process causing it is situated in the nuclear and infranuclear portion of the pathway. In the majority of cases of this type, the strabismus is due to paralysis of individual muscles and is usually readily diagnosed. In others it is due to the presence of contractures and fibrous bands, as in the Duane syndrome, while in some it is undoubtedly due to faulty anatomical relationships, such as abnormal insertions of muscles or anomalies in position of certain muscles. A common example of the latter is an abnormal position of the inferior oblique in its relation to the lateral rectus muscle. The inferior oblique is found beneath the lateral rectus muscle, close to where this muscle becomes tendinous, and seems to be bound with the muscle in a common sheath. If this relation is not recognized, the inferior oblique may easily be picked up with the lateral rectus muscle on the muscle hook when a resection of this muscle is planned. It is advisable to search for the inferior oblique and make sure it is in its normal position before proceeding with a surgical procedure on any lateral rectus muscle.

The part played by abnormal check ligaments, muscle foot-plates, etc., is difficult to evaluate because, in my experience, it is not possible to differentiate between "abnormal" and normal check ligaments in the usual operative procedure. It seems more plausible to consider any abnormalities of the ligamentous expansions and contractures in muscles as the result of long-standing strabismus, rather than the cause. Cases of incomitant strabismus should be recognized, since treatment of this condition is generally different from that of comitant strabismus. Unfortunately, the diagnosis is not always easy, since patients with ocular muscle paralyses of long standing do develop comitance, but a careful examination will usually reveal unmistakable signs of remaining incomitance.

Lesions of the supranuclear pathway for versions will result in a disability of both eyes to move equally and simultaneously in one or more directions of gaze. In Figure 2 a supranuclear lesion situated in the pathways for voluntary dextroversion

will produce an equal embarrassment of the two eyes in willed movements to the right. There will be no strabismus, since muscles of the two eyes will be affected which move them equally and simultaneously in one direction.

In comitant strabismus it is apparent that we are dealing with lesions situated in the supranuclear mechanism having to do with vergences and not with versions, and we must look therefore into the vergence mechanisms for the seat of the difficulty.

Comitant strabismus is always innervational in origin. This is inherent in the very nature of comitance and is proved by the fact that the angle of deviation of the visual axes disappears when the patient is under general anesthesia. Some pathologic process has disturbed the normal vergence mechanism which lies above the level of the nuclei of the oculomotor nerves; it is therefore a supranuclear lesion. In con-

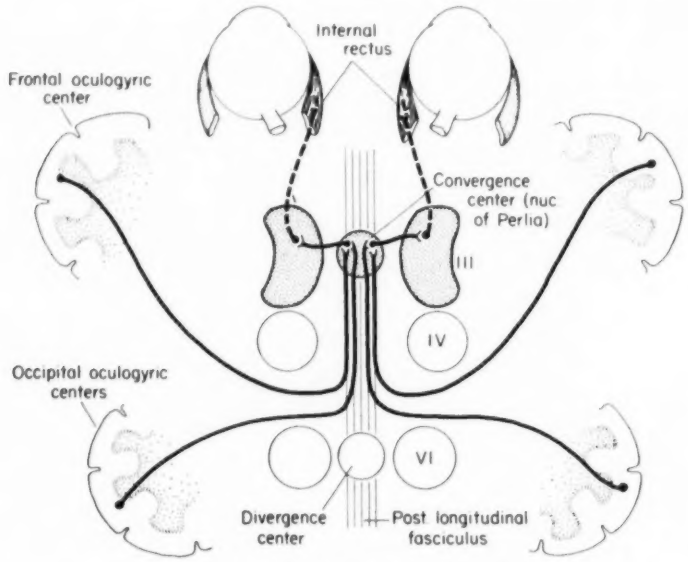


Fig. 3.—Mechanism for convergence.

vergent comitant strabismus we are dealing with an alteration in the normal amount of convergence innervation. Either the convergence mechanism is sending an excessive innervation to both medial rectus muscles, or the divergence mechanism, if one exists, is sending an insufficient innervation to the lateral rectus muscles for any particular degree of vergence required.

The mechanism, or subcortical center (so-called), for convergence is thought to be Perlia's nucleus, as shown in Figure 3. Convergence is largely a reflex act, and although willed convergence can be brought about by many persons, no convergence has ever been demonstrated in experimental animals or in man by electrical stimulation of the frontal oculogyric centers. The existence of a divergence mechanism is disputed. Figure 4 shows a hypothetical divergence mechanism. Some authors claim that divergence of the visual axes is brought about solely by an inherent elasticity of the lateral rectus muscles and the orbital fascia, with relaxation of the

convergence tonus to the medial rectus muscles. As stated by Scobee,[1] "The 'force' of convergence pulling against the 'spring' of elasticity inherent in the orbital structures and tending toward divergence satisfies the requirement of an antagonist for the convergence innervation." Other authors, however, believe that a factor in divergence is an active innervation to the lateral rectus muscles, in addition to the relaxation of the medial rectus muscles. These authors believe that divergence paralysis, and likewise intermittent exotropia, can be explained only on this basis. Evidence is accumulating which suggests that the latter view is correct, and that divergence is an active innervation to the lateral rectus muscles, and not due merely to their elastic pull and that of other orbital tissues in the absence of active convergence tonus.

Henderson and Crosby [2] (Fig. 5) have found divergence movements on stimulating certain regions in the oculogyric centers in monkeys. These movements could

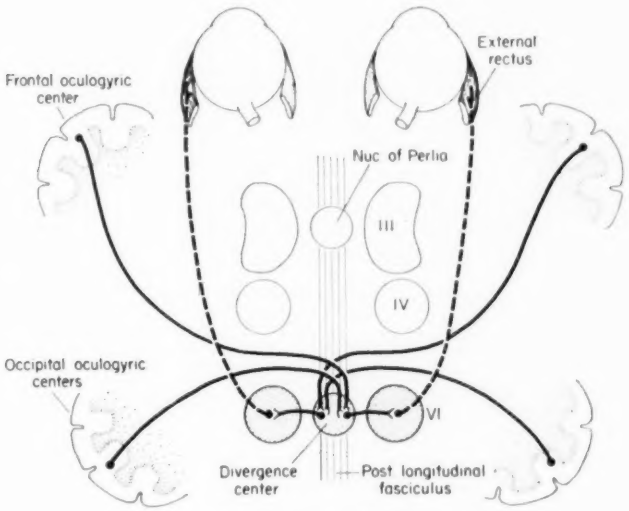


Fig. 4.—Mechanism for divergence.

be brought about as a result of inhibition of convergence and do not prove, therefore, that active divergence exists; but experiments carried out by Dr. Yasuna, Dr. Nulsen, and myself, with the surgical help of Dr. Scheie and Dr. Ojers, prove that the lateral rectus muscles are actively innervated during divergence. Using insulated bipolar electrodes inserted into one of our lateral rectus muscles, we recorded the changes in electrical potential and the number of impulses discharged per second in this muscle in varying positions of gaze. Figure 6 shows the changes in number of impulses per second in turning the gaze from the primary to a secondary position. In diverging from the convergent position, there is a burst of impulses which pre-

1. Scobee, R. G.: The Oculatory Muscles, Ed. 2, St. Louis, C. V. Mosby Company, 1952, p. 164.

2. Henderson, J. W., and Crosby, E. C.: An Experimental Study of Optokinetic Responses, A. M. A. Arch. Ophth. **47**:43, 1952.

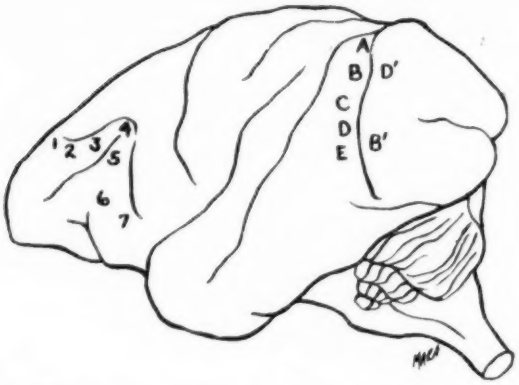


Fig. 5.—Left hemisphere of Macacus mulattus. Letters *A* through *E* indicate the localization pattern of conjugate movements on the preoccipital eye field (Area 19), as follows: *A*, deviation upward; *B*, eyes obliquely upward to right; *C*, eyes to right; *D*, eyes obliquely downward to right; *E*, eyes downward. *D'* and *B'* represent conjugate movements from the occipital eye field (Area 18), as follows: *D'*, eyes obliquely downward to right; *B'*, eyes obliquely upward to right.

Numbers *1* through *7* represent localization areas on the frontal eye field, as follows: *1* and *7*, conjugate ocular deviation obliquely downward to right; *2* and *6*, divergence; *3* and *5*, conjugate right horizontal deviation; *4*, conjugate deviation upward to right.

Note duplication of pattern on the frontal eye field.

From Henderson and Crosby.[2]

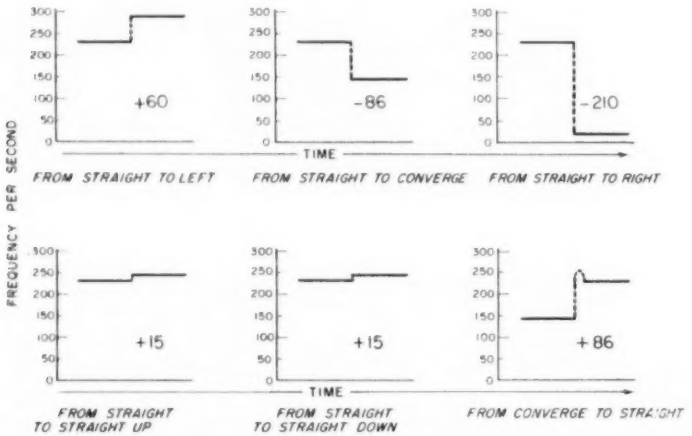


Fig. 6.—Change in number of impulses per second from the left lateral rectus muscle when the eye is turned from the primary position. In the primary position approximately 230 impulses per second were recorded. Because of the slowness of the kymograph, the change in rate could not be recorded. This change is indicated by dotted lines. It was evident, however, that in movements of divergence a burst of impulses occurred in excess of 230, representing the basic tonus in the primary position (see lower right-hand graph).

cedes the actual movement of the eye by some milliseconds, showing that this electrical activity produces and is not the cause of the eye movement. This discharge is shown in Figure 7, one of the actual records. Figure 8 shows a plotting of change in electrical potential and number of impulses discharged per second, which gives a better idea of the change in activity in the muscle in different positions of gaze than a graph of the number of impulses per second alone. An interesting feature is the activity that occurs in the lateral rectus muscles during vertical changes in

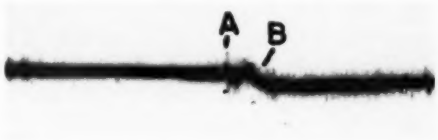
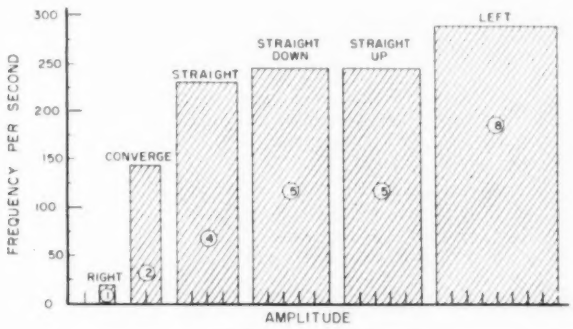


Fig. 7.—Electrical activity from the left lateral rectus. On the command to diverge, at *A*, a burst of impulses is seen, which precedes the actual movement of the globe, shown by the downward displacement of the record at *B*.



	f/sec	amplitude
RIGHT	20	1
CONVERGE	144	2
STRAIGHT	230	4
STRAIGHT DOWN	245	5
STRAIGHT UP	245	5
LEFT	290	8

Fig. 8.—Bar graph of the electrical activity recorded from the left lateral rectus muscle, in terms of frequency per second and amplitude of impulses when the eye is moved in different positions of gaze.

position, probably for the purpose of steadying the globe in its upward and downward movement by cocontraction of the lateral and medial rectus muscles of each eye.

These experiments prove that during divergence from the convergent position the lateral rectus muscles are actively innervated. Divergence is due, therefore, to cocontraction of the lateral rectus muscles, with simultaneous inhibition of the medial rectus muscles. A divergence mechanism, therefore, exists.

As postulated above, comitant esotropia must be due either to excessive innervation to the medial rectus muscles from the convergence mechanism or to insufficient innervation to the lateral rectus muscles from the divergence mechanism. Any pathologic process which so changes the effectiveness of either of these mechanisms is likely to produce comitant esotropia, if not held in check or neutralized by some other mechanism.

In October, 1952, I summarized our knowledge of the physiology of the neuromuscular mechanism which keeps the normal eyes in alignment in a symposium held at the annual meeting of the American Academy of Ophthalmology and Otolaryngology (to be published in the Transactions of the Academy). It was pointed out that the reflex control of ocular movement is the important force performing this function and that, together with the anatomic factors, which, of course, influence to a considerable degree the position of the visual axes in relation to each other, two groups of tonic impulses keep the eyes in alignment. These are (1) the postural reflexes, as shown in Table 1, and the optomotor reflexes, as shown in Table 2. The anatomic basis for these reflexes is diagrammed in Figure 9.

TABLE 1.—*Postural Reflexes*

1. Semicircular canals
2. Otolith apparatus
3. Stretch reflexes from neck muscles
4. Proprioception from ocular muscles
5. Tonic convergence

TABLE 2.—*Optomotor Reflexes*

1. Fixation reflex
2. Following reflex
3. Fusional reflex
4. Accommodative convergence

In the normal person the anatomic factors, the postural reflexes, and the optomotor reflexes keep the eyes in good alignment in all positions of gaze. Small changes in either the anatomic factors or the postural reflexes may be compensated by the remaining two. Failure of the optomotor reflexes alone never produces a deviation of the visual axes. The optomotor reflexes can act as a check or a preventive to the development of strabismus, but their absence can never be a cause of strabismus.

We have outlined the various sites in the neuromuscular mechanism for eye movements where pathologic processes could produce both incomitant and comitant strabismus. We may now make a brief inquiry how pathologic processes disturb the vergence mechanisms and produce comitant strabismus (Table 3).

The tonic convergence of childhood is normally excessive. Comitant esotropia generally follows the loss or impairment of sight in one eye in childhood. As soon as the image of the poor eye fails to elicit an optomotor fusional response, the excessive tonic convergence makes itself manifest by a turning in of the poor eye. Strabismus, therefore, may be due to a sensory obstacle. In most cases esotropia will be produced, but occasionally exotropia appears. Refractive errors produce strabis-

mus by adding the effect of accommodative convergence for distance in the hyperope or the lack of it for near vision in the myope to the tonic convergence tonus. Esotropia due to hyperopia is much commoner than exotropia due to myopia. Slight paralysis of vertically acting muscles sufficient to dissociate the eyes in childhood and make fusion impossible may release the excessive tonic convergence and produce comitant esotropia. It has been shown that both alcohol and anoxia cause an increase in convergence tonus for distance.[3] Under experimental conditions, a person with an esophoria may be made esotropic by the lack of oxygen. It seems likely that the mechanism of this change resides in eliminating an inhibitory influence of the cortex on the convergence center. The cortex normally inhibits the activity of most subcortical centers, and it is likely that the convergence mechanism is kept

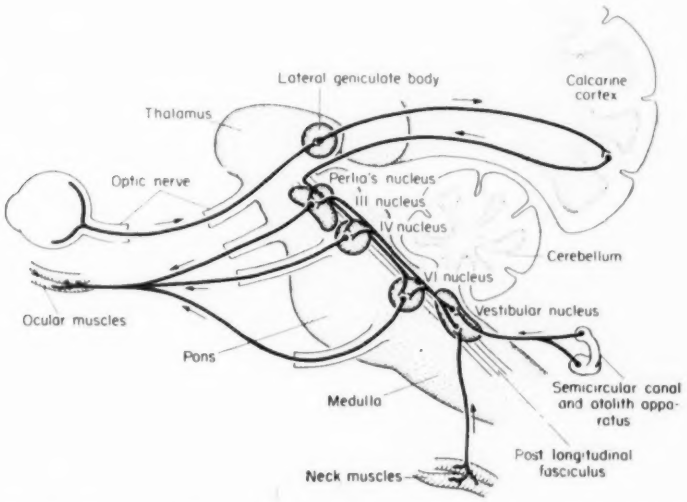


Fig. 9.—Diagram of the postural and optomotor reflexes to the ocular muscles.

TABLE 3.—*Convergence and Divergence Mechanisms*

Convergence	Divergence
1. Tonic convergence of childhood (*a*) Sensory obstacle (*b*) Vertical muscle palsy	1. Excessive divergence impulses in childhood giving rise to intermittent exotropia
2. Accommodative convergence	2. Paralysis of divergence mechanism, giving rise to syndrome of divergence paralysis
3. Release of cortical inhibition	

under cortical inhibition throughout life. Anoxia depresses cortical activity and so releases excessive convergence tonus.

On the side of the divergence mechanism, blocking the normal divergence impulses to the lateral rectus muscles will result in convergent strabismus, and excessive divergence impulses will cause divergent strabismus. The latter is probably the explanation of cases of intermittent exotropia, in which neither sensory obstacles

3. Adler, F. H.: Effect of Anoxia on Heterophoria and Its Analogy with Convergent Concomitant Squint, Arch. Ophth. **34**:227, 1945.

nor refractive errors have anything to do with the production of the strabismus. This condition always starts as a divergence excess, and at first the convergence power is unimpaired.

It is well known that in many cases of comitant esotropia in very young children one or generally both eyes show a weakness in abduction; i.e., on dextroversion the right eye fails to turn out as far as it should, and on levoversion the left eye does similarly. These patients do not have paralyses of their lateral rectus muscles, for if the adducted eye is covered the open eye can abduct fully. Occasionally it is necessary to patch one eye for a week or so in order to reestablish abduction. I have had the opportunity to see several cases of intermittent esotropia in babies in whom there was no demonstrable deviation of the visual axes in the primary position while in the office, and yet they showed this bilateral insufficiency of abduction in versions. The abduction of each eye used alone was normal. In some cases this may be the first step in the development of comitant esotropia, and it appears that an interference with the normal divergence innervation was the causative factor. This situation is somewhat comparable to that found in cases of divergence paralysis occurring in adults. In this condition there is an esotropia for distance, and for any one distance the angle of deviation remains the same in versions. For near, the eyes are in alignment, differentiating this condition from comitant esotropia in children, but this difference may be due to the age at which the condition first appears. In adult life the excessive tonic convergence tonus has disappeared and the accommodative convergence relationship has become established by usage. In the infant the excessive tonic convergence tonus may cause an esotropia for near once the eyes have been sensorially dissociated by weakness of divergence. It has recently been shown that divergence paralysis can occur in the adult in various conditions which have nothing in common save elevated intracranial pressure.[4] This indicates that the pathways serving divergence must be exposed to increased intracranial pressure and are rather vulnerable. This could well apply to the infant as a result of birth trauma.

A large proportion of children born with cerebral palsy have esotropia. Most of these children show not only evidence of individual ocular muscle paralyses, but signs of gaze paralyses, with an intensification of end-position nystagmus in either dextroversion or levoversion—in other words, signs of a combination of supranuclear and nuclear damage. Some children with esotropia but with no obvious signs of cerebral palsy show evidence of damage to cortical pathways, indicating either damage to the brain or lack of myelination. Many of these children have reading difficulties, mirror writing, and personality problems.

SUMMARY

Frank incomitance in a case of strabismus localizes the disturbance to the nuclear and infranuclear pathways. Although this is generally due to a defect in innervation, incomitance due to the presence of fibrous bands and to faulty anatomic relations does occur.

Comitance is always a sign of involvement of the supranuclear mechanism for vergences, and all truly comitant squint is innervational in origin. This type of

4. Chamlin, M., and Davidoff, L. M.: Divergence Paralysis with Increased Intracranial Pressure, A. M. A. Arch. Ophth. **46**:145, 1951.

strabismus can be due to a disturbance in either the convergence or the divergence mechanism. For the existence of the latter I have offered physiologic evidence. Some etiologic factors which produce comitant esotropia are known, such as the loss of the optomotor reflexes in early life, at a time when tonic convergence is excessive, and the presence of hyperopia, imposing an accommodative convergence on the tonic convergence of childhood. Other forms of comitant esotropia occur about which as yet we know little. Abolition of cortical inhibition probably can cause this esotropia in some cases, since one can produce it experimentally by alcohol and anoxia. Failure of development of nerve tracts or lesions in either the convergence or the divergence mechanism can account for a group of cases which may be basically allied to cases of divergence paralysis.

MINIMAL AMOUNT OF X-RAY EXPOSURE CAUSING LENS OPACITIES IN THE HUMAN EYE

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CURRENT events have focused attention on the ocular effects of radiation, and prime interest has centered about cataract production. A considerable amount of experimental work has shown that x-radiation of less than 250 r (200 to 1,000 kv.) can cause lens opacities in animals [1] and that the younger the subject the greater the susceptibility. There is, further, evidence of a species variation by a factor of 2 to 3. The susceptibility of man is, of course, much more difficult to ascertain, and reliable data on this all-important aspect are limited to a few isolated instances. Our aim in the present study was to contact and examine as many persons as possible whose eyes had been exposed to a known amount of x-radiation and to determine thereby the least exposure which could cause an opacification of the lens that was visible with the ophthalmoscope or the slit-lamp biomicroscope.

Similar studies have been attempted in the past, the most comprehensive reports being those of Rohrschneider,[2] Desjardins,[3] and Poppe.[4] However, the patients listed in these reports were, for the most part, irradiated prior to 1930, when dosimetry was recorded in skin erythema units. Since insufficient data were given as to energy, filtration, and other pertinent factors, it is impossible to estimate the amount of x-radiation received by the lens. This insufficiency of data was emphasized by Poppe, who as late as 1942 concluded that the minimal cataractogenic dose, expressed in roentgens, was not known for human beings. However, it is pertinent to note that Rohrschneider [2] concluded that the cataractogenic dose was of the same order of magnitude as the skin erythema dose, while Desjardins [3] concluded that the lens was relatively refractory in comparison with other tissues.

Supported in part by a grant from the Atomic Energy Commission.

From the Howe Laboratory of Ophthalmology, Harvard Medical School, and Massachusetts Eye and Ear Infirmary.

1. (*a*) Cogan, D. G., and Donaldson, D. D.: Experimental Radiation Cataracts: I. Cataracts in the Rabbit Following Single X-Ray Exposure, A. M. A. Arch. Ophth. **45**:508, 1951. (*b*) Upton, A. C.; Christenberry, K. W., and Furth, J.: Comparison of Local and Systemic Exposures in Production of Radiation Cataract, ibid. **49**:164, 1953.

2. Rohrschneider, W.: Klinischer Beitrag zur Entstehung und Morphologie der Röntgenstrahlenkatarakt, Klin. Monatsbl. Augenh. **81**:254, 1928.

3. Desjardins, A. U.: Action of Roentgen Rays and Radium on the Eye and Ear: Experimental Data and Clinical Radiotherapy, Am. J. Roentgenol. **26**:639, 1931.

4. Poppe, E.: Experimental Investigations of the Effects of Roentgen Rays on the Eye, Oslo, I Kommisjon hos Jacob Dybwad, 1942.

During the past two decades, since doses have been measured in roentgens, there have been surprisingly few quantitative observations on cataract production by x-rays. Leinfelder and Kerr[5] found cataracts in two patients who had received something in excess of 1,400 r but no cataract in a patient who had received 1,350 r two years previously. Hunt[6] noted that cataracts occurred five years after delivery of 875 r (130 kv.). Schulz and Healy[6a] referred to a lens opacity occurring in an eye which had been exposed to 1,200 r five years previously. In a preliminary report, Merriam[7] stated he had found no cataracts with amounts of x-rays of less than 1,500 r.

MATERIAL

The 40 patients on whom this report is based were irradiated for various reasons.[8] Some had been given the radiation to the eye as part of the effective treatment of lymphomas or basal-cell carcinoma of the ocular adnexa. For these patients the radiation was from a 200 or a 1,200 kv. source, and the amount was determined with the accuracy usual in modern x-ray therapy. Since the treatment was given directly through the eye, the air dose was a reliable indication of the amount of radiation received by the lens.

In another group of patients the eye was irradiated incidentally in the treatment of various dermatoses. For the patients included in this report, shielding of the eye was omitted either because use of the shield was impracticable or because the amount of radiation reaching the eye was thought to be of no consequence. The eye was not centered in the beam during these exposures, and the x-ray energy was only 100 kv. To estimate the amount of radiation which had reached the eye during these exposures, a Victoreen meter was embedded in wax and placed in the orbit of a phantom skull. Measurements were then made during irradiation carried out in a manner similar to that which had been used in the treatment of patients.

In the group of patients with dermatoses there was a degree of selectivity in that all the patients were young adults and the majority were females.

Observation for lens changes were made with the ophthalmoscope or with the slit-lamp biomicroscope. No patient was included who had received the radiation less than one year previously, and for most patients the observation period was several years. The criterion for attributing a lens change to radiation was the finding of the morphologic changes which have been described as characteristic of irradiation; in the tumor cases a further criterion was the absence of the changes in the opposite eye, which was not exposed to appreciable radiation. All patients were eliminated from the series for whom there was any doubt in our minds as to the validity of the observations or the amount of radiation received, and no observations were included on patients who had received more than 3,000 r to the eyes.

RESULTS

The findings are arranged in the accompanying Table according to increasing dose. Within the limitations of lack of uniformity that is necessary in any series of human beings, the following observations seem significant.

No radiation-induced opacity of the lens occurred with doses of 400 r (100 kv.) in observation periods of 6 to 9 years, and no opacity occurred with 350 r (100

5. Leinfelder, P. J., and Kerr, H. D.: Roentgen-Ray Cataract: An Experimental, Clinical, and Microscopic Study, Am. J. Ophth. **19**:739, 1936.

6. Hunt, H. B.: Cancer of the Eyelid Treated by Radiation with Consideration of Irradiation Cataract, Am. J. Roentgenol. **57**:160, 1947.

6a. Schulz, M. D., and Heath, P.: Lymphoma of the Conjunctiva, Radiology **50**:500, 1948.

7. Merriam, G. R., Jr.: A Clinical Report on Radiation Dosages Producing Cataract, Second Conference on Radiation Cataracts, National Research Council, 1950.

8. Drs. Chester N. Frazier, Robert F. Tilley, and Milford D. Schulz assisted in obtaining contacts with the patients.

Data on Forty Patients with Lens Opacities Following X-Irradiation

Case No.	Total Amount of X-Rays Delivered to Eye, in Roentgens*	Energy, Kv.	Time Over Which X-Rays Were Given	Age of Patient at Time of Irradiation; Sex	Condition for Which X-Ray Treatment Was Given	Time Between Irradiation and Examination, Yr.†	Type of Opacity
1	23	100	Single exposure	23; F	Dermatosis	10	None
2	24	100	Single exposure	24; F	Dermatosis	12	None
3	48	100	1 wk.	17; F	Dermatosis	6	None
4	48	100	2 wk.	30; F	Dermatosis	6	None
5	74	100	2 wk.	17; M	Dermatosis	7	None
6	91	100	1 mo.	32; F	Dermatosis	10	None
7	93	100	3 wk.	15; M	Dermatosis	9	None
8	116	100	1½ mo.	30; F	Dermatosis	7	None
9	128	100	1 mo.	16; F	Dermatosis	11	None
10	153	100	1 mo.	20; F	Dermatosis	11	None
11	157	100	1 mo.	20; F	Dermatosis	4½	None
12	178	100	6 wk.	15; M	Dermatosis	10	None
13	194	100	2½ mo.	26; F	Dermatosis	13	None
14	194	100	2½ mo.	17; M	Dermatosis	12	Congenital: peripheral rider opacity in both lenses
15	185	100	1½ mo.	21; F	Dermatosis	4	Congenital: anterior axial embryonal (Y-shaped)
16	232	100	1½ mo.	19; F	Dermatosis	2	None
17	232	100	1½ mo.	27; F	Dermatosis	4½	None
18	232	100	1½ mo.	17; M	Dermatosis	2	None
19	253	100	1½ mo.	25; F	Dermatosis	14	None
20	257	100	1½ mo.	22; F	Dermatosis	4	Congenital: a few punctate white opacities scattered in posterior cortical layers of both eyes
21	280	100	3 mo.	25; M	Dermatosis	11	None
22	294	100	2 mo.	19; M	Dermatosis	10	None
23	343	100	4 mo.	16; F	Dermatosis	9	None
24	353	100	5 mo.	20; M	Dermatosis	13	None
25	365	100	5 mo.	17; F	Dermatosis	11	Congenital: a few small white punctate opacities in adult nucleus
26	365	100	7 mo.	18; M	Dermatosis	11	None
27	365	100	3½ mo.	16; M	Dermatosis	9	Congenital: a few punctate white opacities scattered in lens substance
28	380	100	3½ mo.	16; F	Dermatosis	1 3 12	None
29	380	100	3½ mo.	22; F	Dermatosis	5	Congenital: tiny suture opacity
30	414	100	4½ mo.	16; F	Dermatosis	8	None
31	446	100	5 mo.	19; M	Dermatosis	9	Congenital: 3 very small opacities in periphery of posterior cortex
32	487	100	4½ mo.	24; F	Dermatosis	6	None
33	300	100	Single exposure	57; F	Lymphoma, conjunctival O. D.	1 9 12	None
34	600	200	2 days	48; F	Lymphoma, conjunctival	2½	Posterior "capsular" ‡
35	600	200	2 days	63; F	Lympho-sarcoma, left orbit	3	None
36	600	200	3 days	66; M	Lymphoma, conjunctival	2½	None
37	800	200	1 mo.	38; F	Malignant lymphoma, conjunctiva O. D.	4½	Lens opacities typical for beginning radiation ‡
38	1,000	200	3 wk.	58; F	Lymphosarcoma, lower lid and conjunctiva O. S.	10 (no observation before this)	Lens opacities, as in Case 37 ‡
39	2,400 or perhaps less (patient moved during exposure)	200	Single exposure	70; M	Basal-cell carcinoma, ala nasi	1 4 12	Mature cataract
40	2,400	1,200	10 days	37; F	Lymphosarcoma, left orbit and lacrimal gland	3	Mature cataract

* Corrected to indicate amount received by lens, expressed as dose in air.

† In the cases in which the lens is clear the latent period is given as the longest postirradiation time, whereas in the cases in which a lens opacity was found the latent period is given as the first time that the lens opacity was noted.

‡ See text.

kv.) in observation periods as long as 13 years. However, unequivocal radiation effects occurred in one of three patients who were known to have received 600 r (200 kv.) through the eye and in all the patients who received 800 r (200 kv.) or more.

The one patient in whom a cataract developed after an exposure of 600 r was 66 years old at the time of the irradiation and was being treated for a lymphoma of the conjunctiva of the bulb and lower lid. The energy was 200 kv., and the treatment was given in three intervals on successive days. The patient had been examined by one of us 14 days after completion of the irradiation and again 1½ years afterward. The lens was clear on both these occasions, and there was no other evidence of radiation-induced abnormality (e. g., epilation or erythema). The tumor receded within a few weeks after the irradiation. Approximately two years after the exposure the patient began to note some diminution of vision on the side which was irradiated. Examination, approximately 2½ years after the irradiation, showed that vision in the irradiated eye was 20/100 + 2, improved to 20/80 with the pinhole disk. Except for the lens, the anterior segment was normal. The lens showed a discoid opacity approximately 5 mm. in diameter, which appeared with the slit illumination to be in the line of the posterior capsule. On ophthalmoscopic examination the periphery appeared somewhat denser than the central portion, suggesting a doughnut in shape, as has previously been described in the early stages of radiation cataract; there was also an indistinct split of the opacity into anterior and posterior laminae, which has previously been termed "bivalve splitting." No peripheral opacities or opacities in the anterior portion of the lens were found. The fundus showed nothing abnormal.

The other eye showed no opacity of the lens or other abnormality.

The patient who had received 800 r to the lens also merits special notation. The opacity was not present one year after the irradiation but was present 4½ years afterward. It was a granular opacity, measuring approximately 2 mm., in the line of the posterior capsule, at the posterior pole, and was unassociated with vacuoles. It had not caused any reduction in visual acuity, as measured by the Snellen test chart.

A similar opacity was found in the lens of the patient who had received 1,000 r, 10 years previously. There was no reason to believe that this had caused any reduction in visual acuity, although the incidental presence of corneal opacities made measurements unreliable. In neither case was there vacuolation that might suggest progression. On the other hand, the two patients who had received somewhat more than 2,000 r had mature cataracts when examined 1½ and 3 years, respectively, after the irradiation.

From the present series, therefore, one would conclude that 600 r was the threshold dose for radiation-induced lens opacities in the human being. This conclusion is, of course, subject to the reservation that opacities may occur with lower doses after a longer period of observation than that in the present cases. However, it is unlikely that any radiation less than 500 r would be expected to have an effect. Nevertheless, data are needed on patients who had received radiation in the 400 to 800 r range 10 to 20 years previously.[9]

9. Since the table was compiled, an additional case of posterior polar opacity has been brought to our attention. The patient, a 4½-year-old child with a hemangioma, had received a

(Footnote continued on next page)

COMMENT

The series purposely includes a predominance of patients who received amounts of radiation that might be considered too low to be of significance. This selection was prompted by the unexpected finding of radiation-induced changes in the lens in animals given low doses, e.g., 75 r in the case of rabbits [10] and 15 r in the case of mice.[1b] No comparable changes were found in human beings with such low doses. It must be said, however, that the patients were not as regularly followed after the irradiation and that such changes might have occurred and subsequently disappeared. We may conclude, therefore, that if changes do occur in human beings with equally low doses of radiation, they are reversible.

The lens opacities occurring in the patients who had received 600, 800, and 1,000 r, developing two or more years after the irradiation, were roughly comparable to the opacity in rabbits following irradiation with 500 r and coming on approximately 100 days after the irradiation. This similarity is, of course, a gross approximation and is not intended to imply a greater susceptibility on the part of the rabbit; rather, it is interpreted as showing that the rabbit lens and the human lens have the same order of susceptibility. It does appear, however, that the latent period is several times as long in the case of human beings as in the case of rabbits.

Finally, it may be noted that the minimal dose found in the present series is in keeping with the threshold values of Hunt,[6] based on observations on two patients. The earlier conclusion that the cataractogenic dose and the skin erythema dose were similar may be expected to hold only when energies of 200 kv. are used. Energies of less than 200 kv. would be expected to be cataractogenic only when the doses are greater than that necessary to cause the skin erythema, whereas energies greater than 200 kv. would be expected to be cataractogenic with doses less than the skin erythema dose.

CONCLUSIONS

Lens opacities were not found in the present series of patients who had received 400 r (energies of 100 kv.) or less to the eye. Lens opacities of definitely radiation type were found in one of three patients who had received 600 r, in the one patient who had received 800 r, and in the one who had received 1,000 r. The time of onset of lens changes after the irradiation in these three patients was 2½ years for the first, between 1½ and 4½ years for the second, and some time less than 10 years for the third. In the two patients who had received 2,400 r, mature cataracts had developed one year four months and three years, respectively, after the irradiation.

While the threshold dose for the production of lens opacities with the energies ordinarily employed (100 to 200 kv.) appears to be of the order of 600 r, more observations are needed in the long-range follow-up of patients whose eyes have been exposed to x-rays in the dosage level of 400 to 1,000 r.

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total of 900 r (50 to 125 kv.) to the region of the inner canthus when he was between 3 and 18 months of age. The eye was believed to have been partially shielded at the time; so the total amount of radiation received by the lens is unknown. The case is therefore of little value for our study other than to confirm the impression that posterior polar opacities can be induced by radiation with doses less than 900 r.

10. Cogan, D. G.; Donaldson, D. D.; Goff, J. L., and Graves, E.: A. M. A. Arch. Ophth. to be published.

CONGENITAL STATIONARY NIGHT BLINDNESS WITHOUT OPHTHALMOSCOPIC OR OTHER ABNORMALITIES

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PRIMARY chorioretinal aberrations with night blindness was the subject of a symposium [1] at the 1949 meeting of the American Academy of Ophthalmology and Otolaryngology. The first abnormality considered in that symposium, and the subject of this paper, was characterized by one of us (F. D. C.) as night blindness present at birth, stationary, occurring in an approximately equal number of males and females, and with no associated abnormalities. Bell [2] collected 19 pedigrees of this disease.[3] A few sporadic cases have probably been encountered in the examination of candidates for the air force in this country [4] and in Canada,[5] and Langdon,[6] in 1915, reported the cases of a father and daughter in Philadelphia who had a reduced light sense and normal fundi. Except for Langdon's observation, however, we believe that the following report is the first to record a family affected typically with this abnormality in North or South America.[7]

Read at the Fifty-Eighth Annual Meeting of the American Ophthalmological Society, Hot Springs, Va., June 6, 1952.

From the Department of Ophthalmology, Columbia University College of Physicians and Surgeons, and the Institute of Ophthalmology, Presbyterian Hospital (Dr. Carroll), and the Department of Physiology and Pharmacology, New York Medical College, Flower and Fifth Avenue Hospitals (Dr. Haig).

1. Leinfelder, P. J.; Carroll, F. D.; Falls, H. F.; Cogan, D. G.; Fralick, F. B., and Duke-Elder, W. S.: Symposium: Primary Chorioretinal Aberrations with Night Blindness; Classification, Tr. Am. Acad. Ophth. **54**:607, 1950.

2. Bell, J.: Treasury of Human Inheritance, London, Cambridge University Press, 1922, Vol. 2, Pt. 1, p. 29.

3. Riddell, W. J. B.: A Pedigree of Hereditary Stationary Sex-Linked Night Blindness, Ann. Eugenics **10**:326, 1940; Two Clinical Tests for Night Blindness, Tr. Ophth. Soc. U. Kingdom **60**:181, 1940. Gates, R. R.: Human Genetics, New York, The Macmillan Company, 1946, Vol. 1, p. 221. Truc, H.: Opin un ancien foyer d'héméralopia nongarienne, Arch. opht. **42**: 481, 1925. Sinclair, W. W.: Hereditary Congenital Night Blindness Without Visible Ophthalmoscopic Changes, Ophth. Rev. **24**:255, 1905. Hudson, A.: Notes on a Case of Congenital Night Blindness, South African M. Rec. **1**:73, 1903.

4. McDonald, R.: Personal communication to the authors.

5. McCullough, C.: Personal communication to the authors.

6. Langdon, H. M.: Hereditary Deficiency of the Light Sense in Otherwise Healthy Eyes, with Report of a Case, Ann. Ophth. & Otol. **24**:469, 1915.

7. Bordley, J. J.: A Family of Hemeralopes, Bull. Johns Hopkins Hosp. **19**:278, 1908. Newman, H. H.: Five Generations of Congenital Stationary Night Blindness in an American Family, J. Genetics **3**:25-28, 1913.

In France this disease is called the "Nougaret type of night blindness." Jean Nougaret (1637-1719) is thought to have introduced it into the parish at Vendémian, near Montpellier, about 300 years ago. The records of 9 generations of the Nougaret family are available,[8] and some members of the 10th generation have been reported by French oculists.[9] In 1831 a young Frenchman who was drafted for military service claimed exemption because of night blindness. The military surgeon in Montpellier found that the draftee could see by candlelight and stated that he was malingering. His exemption was denied, and he served for seven years. Later he was examined again and at this time stated that his father, grandfather, and great-grandfather were unable to see at night. This time he was believed and was exempted from further service. From an intensive search of parish records it was

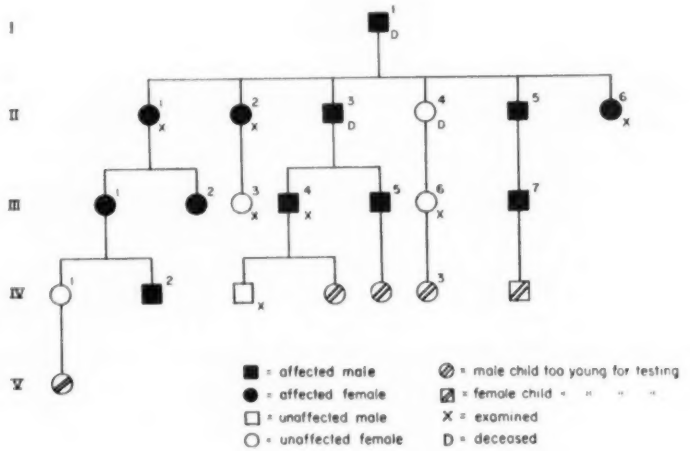

Fig. 1.—Pedigree chart.

concluded that a butcher, Jean Nougaret, born about 1637, was the first person in the locality with night blindness, and he is known as the founder of the genealogical line, which includes over 2,000 members, of whom 135 are said to have had night blindness. This pedigree, which is one of the longest on record in human genetics, reveals a genetic fact of practical clinical significance; i. e., an unaffected child of parents with this type of night blindness never carries the disease, and none of the descendants of such a child are involved.

HISTORY OF AN AMERICAN FAMILY AFFECTED WITH CONGENITAL STATIONARY NIGHT BLINDNESS

A physician's wife (Fig. 1, *II-6*), aged 53, came to the office of one of us (F. D. C.) for a refraction. She mentioned incidentally that she had had night blindness since birth and stated on being questioned that numerous other members of her family (including her father, then dead)

8. Nettleship, E.: A History of Congenital Stationary Night Blindness in 9 Consecutive Generations, Tr. Ophth. Soc. U. Kingdom **27**:269, 1907.

9. Dejean, C., and Gassenc, R.: Note sur la généalogie de la famille Nougaret de Vendémian, Bull. Soc. opht. France **1**:96-100, 1949. Jayle, G. E., and Ourgaud, A.: La vision nocturne et ses troubles, Paris, Masson & Cie, 1950.

were similarly affected. With a correction of + 0.75 sph., visual acuity was 20/20 in each eye. Ophthalmoscopic examinaton revealed nothing abnormal. The visual fields, as examined with an illumination of approximately 7 foot-candles, were normal.

Subsequently we examined two of this woman's sisters (Fig. 1, *II-1, II-2*), two of her nieces *(III-3, III-6)*, a nephew *(III-4)*, and a grandnephew *(IV-2)*. Of these seven members of the family examined, all had normal fundi, four had night blindness, and three had normal dark adaptation. Other members of the same or later generations were dead or lived too far away to be available for examination, but whether or not a relative was night-blind appeared to be well known to his immediate family, and the family history, verified by each adult examined, is believed to be reliable.

Sorsby [10] stated that a pedigree shows autosomal dominant inheritance if the following criteria are satisfied: (*a*) direct transmission over three generations; (*b*) a frequency of affected members approximating 50% of the total number concerned, and (*c*) no predilection for either sex. The present pedigree satisfies these criteria by showing the condition to have extended over four generations and to have affected 12 of 17 members concerned, 7 of them males and 5 females; it was transmitted by either males or females.

Clinical Findings.—The physician's wife (Fig. 1, *II-6*) was intelligent and willing to demonstrate for us the extent of her visual capacity. She said she liked to drive her automobile at night. On an overcast night one of us rode with her, at first with some trepidation. She drove without difficulty; and when she was questioned about objects beside the road but outside the direct range of the headlights, her responses indicated that she could see them as well as the observer, who had normal dark adaptation. She refused to back the car in the dark, however, and needed a flashlight to walk from the garage to her house, although there was sufficient light from the overcast sky for the normal observer to see his way. "Just give me a flashlight," she said, "and I can see as well as anybody." Under the ordinary illumination of her home she had no visual difficulty; but when we walked on an unlighted terrace, her vision was considerably less acute than her observer's and, except for the first few minutes, did not improve in the dark. The moon was hidden by clouds, but the observer could easily see the steps, the edge of a stone terrace, a few chairs; the patient saw none of these things and did not see her cat walking along a few feet away. She said that she understood her limitations and that this poor illumination always made her uneasy and nervous. She said she refused to attend outdoor dinner parties in the evening because she could not see the people with whom she was eating and talking and often failed to see the outstretched hand of a friend who came to greet her.

With a Macbeth illuminometer,[11] one of us (F. D. C.) on another evening tried to determine at what level of illumination the patient first seemed to have visual impairment. Under a street light the illumination was approximately 0.1 f.-c. She appeared to have normal vision there and also at a distance from the street light at which the reading was 0.01 f.-c., but between this point and the point at which the reading was 0.001 f.-c. she began to show visual impairment, and below an illumination of 0.001 f.-c. she seemed unable to distinguish anything. This rough test tended to confirm the accurate laboratory tests with the Hecht-Shlaer adaptometer, reported in the Appendix.

The patient's nephew (Fig. 1, *III-4*), 30 years of age at the time of writing, stated that he had noticed no trouble with night vision until he was 12 years old, when he first left a well-lighted large city and went to a summer camp. Before retiring at night, the boys walked from their tents to a nearby lavatory. He began to think he could not see as well as the other boys and became sure of it one night when he plunged off a boardwalk into a cesspool. His father (Fig. 1, *II-3*), now dead, was notified of the boy's difficulty and told the boy at this time that he also was night-blind. The youngster learned to take the arm of a friend at night when necessary. Occasion-

10. Sorsby, A.: Genetics in Ophthalmology, St. Louis, C. V. Mosby Company, 1951, p. 7.

11. Dr. LeGrand Hardy, Dr. Gertrude Rand, and Miss Catherine Rittler, of the Arnold Knapp Laboratory of Physiological Optics, Institute of Ophthalmology, New York, assisted in this phase of the study.

ally the other boys, recognizing his handicap, played pranks on him. One night, when he was paddling in the bow of a canoe, one of his companions said the canoe was about to touch the shore and yelled to him to jump out; he did and found himself far out in the lake. When he grew older and attended college, he had difficulty at night walking across the poorly illuminated campus and once struck a post with great force. Gradually he learned the footpaths well and when in doubt walked with his arms stretched out in front of him, one crossed over the other. Now employed as a chemist, he frequently travels about the country for his company. Always with him is a pencil flashlight. At night he drives his car and often visits friends with his wife, but he says he usually takes her arm going down outside steps unless he uses the flashlight.

In wartime people with this abnormality are more seriously handicapped. During World War II the physician's wife (Fig. 1, *II-6*) was a volunteer driver for the Red Cross in her community. During the practice blackouts the drivers had to sit in the dark for 20 minutes before going outdoors. She found she could not see any better after 20 minutes than after the first few

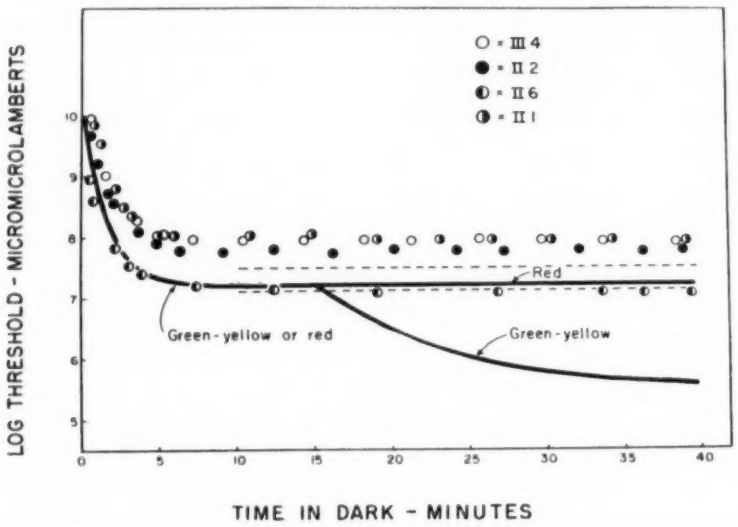

Fig. 2.—The time course of dark adaptation at 7 degrees from the foveal center in the nasal retina following a three-minute exposure to a white light of 6,000 millilamberts intensity. The curves are from averaged data of three expert observers for which tests lights were red and green-yellow, as indicated. The lines of dashes are the upper and lower limits of the normal cone plateau (110 inexperienced subjects). The circles are individual determinations with green-yellow test light on four patients identified in Figure 1, as indicated.

minutes and, unlike the other drivers, was totally unable to drive with parking lights during dim-out or black-out. Her niece (Fig. 1, *III-2*) was in the Women's Auxiliary Corps of the Army and on night maneuvers once fell into a ditch. Her nephew (Fig. 1, *III-4*) volunteered for the Army Air Corps and served for a short time in this country. On guard duty at night he had great visual difficulty. Finally, he told his officer about his night blindness and, after a confirmatory examination, was discharged.

Dark-adaptation measurements, made by one of us (C. H.) and described in the Appendix, indicated that all four night-blind patients examined were totally lacking in rod function (Figs. 2, 3, and 4). One subject had completely normal cone function, and the other three had cone thresholds 0.20 and 0.40 log units above the highest normal; this is not a large deviation from normal, and these three subjects had had no previous experience with dark-adaptation tests. To

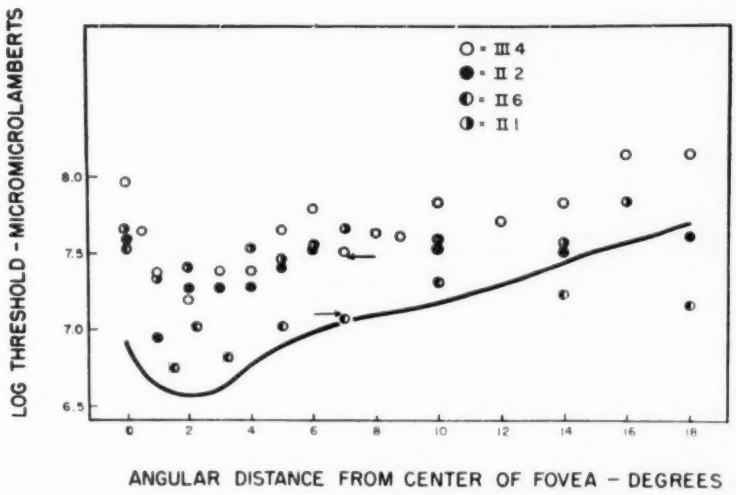

Fig. 3.—Absolute-threshold contours in the temporal retina of four night-blind patients (circles) and averaged cone-plateau contours of three normal subjects (curve). The circles are individual determinations with green-yellow light and the eye completely dark-adapted. The curve represents the averaged cone-plateau thresholds for red light for three expert observers, and the arrows show the limits for 110 normal subjects at the 7-degree locus nasal to the foveal center.

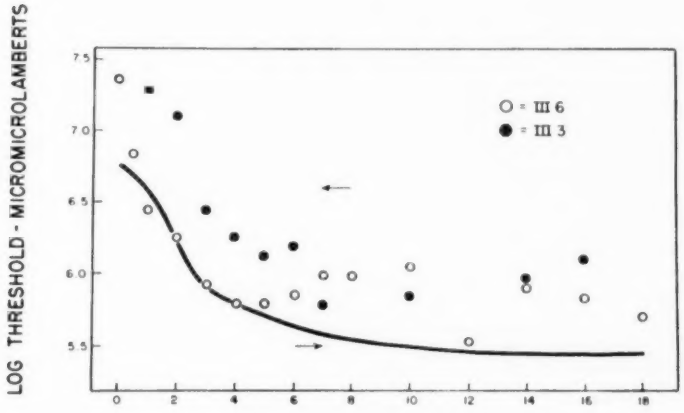

Fig. 4.—Absolute-threshold contours in the temporal retina of the normal members of the family (circles) determined with green-yellow test light. The arrows are at the upper and lower limits for 110 inexperienced normal subjects at 7 degrees nasal to the foveal center.

date, no histological specimens have been available for examination, so that it is not possible to state whether or not nonfunctional rods are present in the night-blind retinas.

The Purkinje phenomenon is the name given to Purkinje's observation, in 1823,[12] that in poor light blue appears brighter than red and that in good light red appears brighter than blue. This phenomenon depends on a shift from rod to cone activity and hence can occur only in regions of the retina where cones and rods are coexistent.[13] A test of this shift in activity was performed as follows[14]: Contiguous patches of Heidelberg red and blue paper measuring

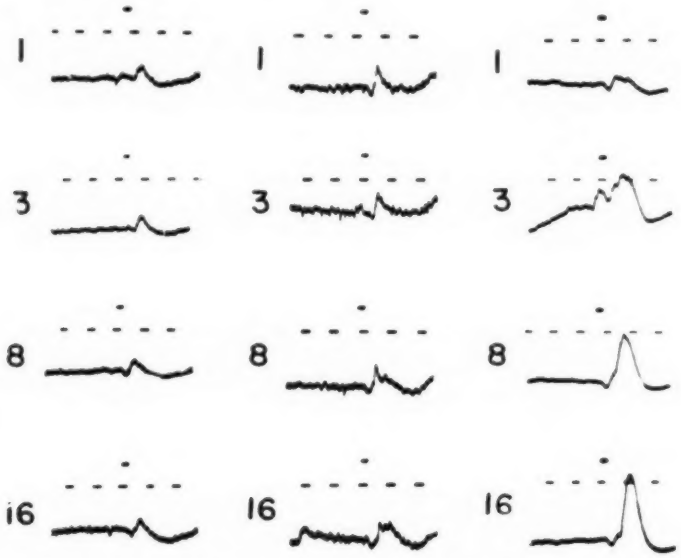

Fig. 5.—The curve at the left is the record of Patient *II-6;* the center curve, the record of Patient *II-2* (given a flash five times the intensity of the other patient); the curve on the right, the record of the normal subject given a flash of the same intensity as Patient *II-6*. The short line (-) indicates that the shutter delivered a flash of 0.025 second at this instant; the time line (line of dashes) indicates 1/10 second units; 100 microvolts equals approximately 3.5 mm. A green light about 8 degrees in diameter and centrally fixated was used. The numbers at the left of the curves indicate the number of minutes in the dark after the eyes had been exposed to an adapting light of moderate intensity (about 30 foot-lamberts).

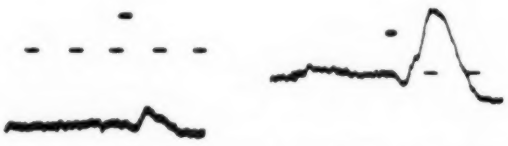

Fig. 6.—The curve at the left is the record of Patient *II-6*, and the curve at the right, the record of a normal subject, both fully dark-adapted and both subjected to the same intensity of flash. The large b-wave is lacking from the response of the patient with pure cone vision.

The symbols have the significance of those in Figure 5.

12. Purkinje, J. E.: Beobachtungen in Versuche zur Physiologie der Sinne, 1823, p. 118.

13. Adler, F. H.: Physiology of the Eye: Clinical Application, St. Louis, C. V. Mosby Company, 1950, p. 645.

14. The tests were performed by Dr. G. Rand, Department of Physiological Optics, Institute of Ophthalmology.

11 by 7½ in. (27.9 by 19 cm.) were placed on a black background. The illuminations, which came from an x-ray viewer, could be varied as desired. To normal eyes blue appears brighter than red when the illumination is below the color threshold, i. e., approximately 0.001 f.-c.; but when the illumination is increased, red appears brighter than blue. Three of our night-blind subjects were tested. To them red always appeared brighter than blue, even in the faintest illumination. This absence of the Purkinje phenomenon is additional evidence of a lack of rod function.

Color vision tests[14] on one patient indicated mildly defective blue-yellow vision and low-normal red-green vision. The results of the battery of tests are shown in appendix II. Refraction (appendix III) showed small to moderate amounts of hyperopia or compound hyperopic astigmatism; the visual acuity with correction was always normal. The visual fields were normal until the illumination was greatly reduced. A contraction of 20 to 40 degrees in the peripheral fields was present when the illumination was between 0.01 and 0.001 f.-c.; below the latter level the patient apparently saw nothing.

Recently, electroretinograms have been recorded on two of these night-blind patients by Prof. Lorrin A. Riggs, of Brown University. Professor Riggs[15] is a pioneer in the use of electroretinography on human subjects, and his findings are of exceptional interest. The technique used was as follows[16]: A contact lens containing the critical electrode was inserted over the right cornea; a neutral electrode was applied to the forehead above the right eye. The difference in potential existing between these two electrodes was amplified and recorded. Figure 5 indicates the electrical response during dark adaptation of eyes which showed no rod function, as compared with the normal. One minute after the beginning of dark adaptation there was little difference in the three responses, but in the normal eye, after three minutes the so-called b-wave had risen markedly and after 28 minutes it had reached a point corresponding to 400 μv. The records for the two patients, on the contrary, show that the b-wave at any time during dark adaptation was very small, if present at all. Since the principal wave of the retinal response reflects primarily the scotopic system of the eye,[15] its absence in these records tends to confirm the impression of a lack of rod-receptor mechanism.[17]

Figure 6 shows the response of a night-blind patient (*II-6*) and a normal subject, both fully dark-adapted. The normal response indicates a negative a-wave, followed by a positive b-wave, whereas the record of the affected subject, with only cone vision, lacks the large b-wave. Complete details of the electrical responses will be published by Professor Riggs.[18]

SUMMARY

A family with congenital stationary night blindness is reported. Dark-adaptation tests, absence of the Purkinje phenomenon, electroretinography, and clinical studies indicated a complete lack of rod function. An unusual opportunity was thus presented for the study of the visual capacity of persons who have normal (or almost normal) cone function only. In peacetime these people are occasionally embarrassed by their handicap, but they marry, have children, and lead almost normal lives; in wartime, however, it is important for the ophthalmologist to recognize the condition, so that those affected will not be so placed that their disability may endanger themselves or others.

15. Riggs, L. A., and Johnson, E. P.: Electrical Responses of the Human Retina, J. Exper. Psychol. **39**:415, 1949. Riggs, L. A.; Berry, R. N., and Wayner, M.: A Comparison of Electrical and Psychophysical Determination of the Spectral Sensitivity of the Human Eye, J. Optic. Soc. America **39**:427, 1949.

16. Johnson, E. P.: Electrical Response of the Human Retina During Dark Adaptation, J. Exper. Psychol. **39**:597, 1949.

17. Riggs, L. A.: Personal communication to the authors.

18. Riggs, L. A.: To be published.

APPENDIX I

Dark-adaptation studies were made upon seven members of the family by means of a modified Hecht-Shlaer adaptometer [19] and a Haig-Lewis adaptometer.[20] Since the experimental conditions differed from those described by Hecht and Mandelbaum [21] in their study of 110 normal subjects, the present measurements require specification. The carriage for the fixation light and the rack on which it rides were replaced by a carriage and rack which permitted changes to be made in the angle of fixation in a matter of seconds. The test field subtended 0.24 degree of visual angle, and the duration of each test flash was 0.2 second. A green-yellow filter (Eastman Wratten filter 73) was used, with maximal transmission at 570 mμ and cut-offs at 550 and 630 mμ. This filter eliminates all blue and violet light below the wave length of 550 mμ, thus avoiding differential absorption of the shorter wave lengths by the yellow macular pigment. This source of error in comparing the functions of the macular and the extramacular region of the retina has been discussed by Wald,[22] Haig and Haig,[23] and Hartridge.[24]

Two experimental procedures were carried out, as follows:

Procedure 1.—The subject was dark-adapted for 30 minutes and then exposed for 3 minutes to a white light of 6,000 millilamberts intensity. During the subsequent dark period the subject fixated, when so instructed, on the dim fixation light, which was so placed that the image of the test flash fell at a locus 7 degrees nasal to the foveal center. Threshold measurements began immediately and were repeated at frequent intervals for 40 minutes or longer. While the threshold was decreasing rapidly, the observer measured it by noting the intensity of a single, just visible flash and the time in the dark at which it was observed.

Later, when plateaux for cone and rod function were reached and the adaptation rate was nil, the threshold was determined more accurately as the intensity which was seen in 9 out of 10 trials. When a green-yellow test light was used, the normal subject showed two phases in dark adaptation (curves, Fig. 2): The first phase was cone adaptation, and the second, beginning at between 12 and 15 minutes, was rod adaptation. The transition from the cone plateau to rod function was marked by a sudden drop in threshold and a loss of green-yellow sensation, the stimulus then appearing white. If a deep-red stimulus (Wratten filter 88) was used, the second, or rod, phase was deferred to 40 or 45 minutes and was extremely shallow (0.20 to 0.30 log unit).[25]

In Figure 2 the curves represent the average data of three expert observers which were obtained with green-yellow and red test stimuli, as indicated. The lines of dashes represent the adjusted extremes of cone plateaux obtained from 110 inexperienced normal subjects by Hecht and Mandelbaum.[21] Since the measurements for these normal subjects were made with violet light, a test field substending 3 degrees, and a different threshold criterion at the plateaux, an

19. Hecht, S., and Shlaer, S.: An Adaptometer for Measuring Human Dark Adaptation, J. Optic. Soc. America **28**:269, 1938.

20. Haig, C., and Lewis, J. M.: Simple Method of Measuring Brightness Threshold of Dark Adapted Eyes at All Ages, Proc. Soc. Exper. Biol. & Med. **41**:415, 1939.

21. Hecht, S., and Mandelbaum, J.: The Relation Between Vitamin A and Dark Adaptation, J. A. M. A. **112**:1910, 1939.

22. Wald, G.: Human Vision and the Spectrum, Science **101**:653, 1945.

23. Haig, C., and Haig, E. M.: Retinal Sensitivity Contours, Abstracts of Communications of XVII th International Physiological Congress, Oxford, 1947.

24. Hartridge, H.: Recent Advances in the Physiology of Vision, Philadelphia, The Blakiston Company, 1950.

25. Hecht, S.; Haig, C., and Chase, A. M.: Influence of Light Adaptation on Subsequent Dark Adaptation of the Eye, J. Gen. Physiol. **20**:831, 1937.

adjustment of their values had to be made for comparison with the present measurements.[26] The circles represent the single measurements of four of the patients when green-yellow test flashes were used.

It is evident that the early data of the patients with night blindness showed the same rate of change of threshold as the normal subjects. Only one patient (Fig. 1, *II-6*), however, had completely normal cones. The other three patients had cone plateaux 0.30 to 0.40 log unit above the highest normal. None of the patients showed any evidence of rod function at this locus, the thresholds remaining at the level of the cone plateau, paralleling the normal curve obtained with a red test light.

In Procedure 1 only one locus of the retina was tested. By means of Procedure 2 many loci, out to 18 degrees in the middle periphery, were examined.

PROCEDURE 2.—After 30 minutes in complete darkness the subject looked at the fixation light placed at 0.1 degree off center, and the intensity of the green-yellow test flash which he saw in 9 out of 10 trials was taken as threshold. The fixation light was then moved in the horizontal meridian to various angles nasally and temporally to as far as 18 degrees and the threshold determined at each position. The natural pupil was used, and in computing threshold intensities appropriate corrections were made for the effect of the oblique pupil upon the retinal illumination.[27] This correction was never more than 0.02 log unit.

The results for the temporal retina are shown in Figures 3 and 4. In order that normal cone function might be compared with the cone function of the night-blind patients, it was necessary to light-adapt the normal subjects and determine the level of the cone plateau, as in Procedure 1, at each locus of the retina. The averaged data for the three expert normal subjects are represented by the curve in Figure 3. It is seen that the curve passes through a minimum at 2 degrees from the foveal center and rises steadily toward the periphery.[28] The patients' data have the same general contour, but those of only one patient (Fig. 1, *II-6*) fall within the normal range at the 7-degree locus (arrows), the greatest deviation being about 0.3 log unit. These results are to be compared with those displayed by two normal subjects under identical conditions (Fig. 4). The thresholds in Figure 4 are all lower than those in Figure 3 and decrease rapidly at first, and then more slowly, toward the periphery. This contour indicates rod function. The data are well within the normal rod range at the 7-degree locus and follow the contour of the averaged thresholds of the three normal observers represented by the curve.

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26. Our measurements (Haig and Haig.[23] Haig, C.: The Intensity Limen in Relation to Rod and Cone Cross-Sectional Areas and Population Densities in Same and Different Retinal Regions, Abstracts of Communications, Eighteenth International Physiological Congress, Copenhagen, 1950) show that this adjustment may be made by adding to the data of Hecht and Mandelbaum log units as follows:

	Log Units		Log Units
Violet and green-yellow		Threshold criterion	
Rod	1.10	Rods	0.20
Cones	0.00	Cones	0.20
3° to 24°		Totals	
Rods	1.8	Rods	3.10
Cones	1.55	Cones	1.75

Since the nasal and temporal threshold contours are symmetrical out to about 10 degrees,[23] these nasal data are valid for both sides of the fovea.

27. Spring, K. H., and Stiles, W. S.: Apparent Shape and Size of the Pupil Viewed Obliquely, Brit. J. Ophth. **32**:347, 1948.

28. Haig and Haig.[23] Haig, C.: The Intensity Limen in Relation to Rod and Cone Cross-Sectional Areas and Population Densities in Same and Different Retinal Regions, Abstracts of Communications of XVIIIth International Physiological Congress, Copenhagen, 1950.

One other normal patient, a boy 5½ years old (Fig. 1, *IV-3*), was tested by a simple method devised for children.[20] His rod threshold was 1.9 log unit, the normal range being 1.6 to 2.5 log units.

In all the adult subjects both eyes were tested nasally and temporally. The results for the nasal retinas and the left eyes were comparable to those reported in this paper for the right temporal retinas.

Adrian, E. D.: Rod and Cone Responses in the Human Eye, Nature, London **154**:361, 1944; Rod and Cone Components in Electrical Response of the Eye, J. Physiol. **105**:24, 1946.

APPENDIX II

Record of Color Tests for Patient II-6

	No. of Errors	Findings
Tests for red-green vision only		
Ishihara (5th edition)	0	Normal
American Optical Company (revised selection)	0	Normal
Bostrom-Kugelberg	0	Normal
Hardy-Rand-Rittler (red-green plates)	0	Normal
Nagel anomaloscope	..	Normal
Tests for red-green and blue-yellow vision		
Hardy-Rand-Rittler (blue-yellow plates)	(1 error)	Mild blue-yellow defect
Farnsworth dichotomy test		Mild blue-yellow defect
Farnsworth-Munsell 100-hue test		Low color discrimination, more marked in blue-yellow region
Color threshold tester (School of Aviation Medicine)		Test passed, but high thresholds for blue and red

APPENDIX III

Refraction of Patients with Night Blindness

O.D.	+0.75 sph.	= 20/20
O.S.	+0.75 sph.	= 20/20
O.D.	+0.75 sph. ⌒	+1.50 cyl., axis 170 = 20/20
O.S.	+1.50 sph. ⌒	+1.25 cyl., axis 30 = 20/20
O.D.	+2.75 sph. ⌒	+0.75 cyl., axis 140 = 20/20
O.S.	+3.25 sph. ⌒	+0.50 cyl., axis 60 = 20/25
O.D.		+0.25 cyl., axis 10 = 20/20
O.S.		+0.50 cyl., axis 165 = 20/20

GLUCOSE UTILIZATION OF THE RETINA

Influence of Various Media

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THE PRESENT investigation was undertaken to determine the glucose utilization of the retina under a variety of physiological and pathological conditions. Because of its unique respiratory activity, the retina occupies a special place among the various tissues of the body. Warburg [1] showed that the retina had the highest rate of respiration and the highest anaerobic metabolism of any tissue studied. Dickens and Simer [2] stated that the aerobic glycolysis in vitro was very high, even greater than that of most tumors. The latter authors concluded that the respiratory quotient was 1, thus implying that the metabolism consisted strictly in oxidation of carbohydrates. As carbohydrates seem to be the main source of energy for the retina, it was thought that an analysis of the glucose utilization of the retina under various experimental conditions offered the possibility of gaining additional information on the metabolism of the retina.

The first essential was to find an optimal medium for glucose utilization by the retina. It was known that the respiratory rate of rat retina was about twice as high in bicarbonate Ringer's solution as in phosphate Ringer's solution.[3] Craig and Beecher [4] observed that the metabolism of rat retina was sensitive to the concentration of the carbon dioxide-bicarbonate buffer system. Santoni [5] noted that glutamic acid increased the respiration of the retina. Ideta [6] observed that increasing the sugar concentration, up to 0.5%, increased the respiratory rate

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This paper is part of a thesis to be submitted by Mrs. Yardeni-Yaron to the Hebrew University in partial fulfillment of the requirements for the degree of Doctor of Philosophy.

1. Warburg, O.: Über die Klassifizierung tierischer Gewebe nach ihrem Stoffwechsel, Biochem. Ztschr. **184**:484, 1927.

2. Dickens, F., and Simer, F.: Metabolism of Normal and Tumor Tissue: II. The Respiratory Quotient, and the Relationship of Respiration to Glycolysis, Biochem. J. **24**:1301, 1930.

3. Krause, A. C., and Sibley, J. A.: Metabolism of the Retina, Arch. Ophth. **36**:328, 1946.

4. Craig, F. N., and Beecher, H. K.: Effect of Carbon Dioxide Tension on Tissue Metabolism (Retina), J. Gen. Physiol. **26**:473, 1943.

5. Santoni, A.: Ulteriori ricerche sul metabolismo della retina: Metabolismo degli aminoacidi, Rass. ital. ottal. **9**:81, 1940; abstracted, Am. J. Ophth. **23**:1290, 1940.

6. Ideta, K.: Studien über die Gewebsatmung der Netzhaut des Kaninchens in Vitro: Einfluss des Zuckers in Serum, Jap. J. M. Sc., III, Biophysics **5**:105, 1938.

and that glycolysis was greatest at a concentration of 0.2 to 0.4%. Oguchi [7] found that the metabolism of rabbit retina was greater in serum than in Ringer's solution. Shaffer, Chang, and Gerard,[8] studying the influence of blood constituents on oxygen consumption of nerve, noted that oxygen consumption of dog retina (one experiment only) was 187% as great in serum as in Ringer's solution. Similar observations were made on brain and nerve. The authors related this increase in respiratory activity largely to the presence of proteins in the serum.

TABLE 1.—*Influence of Tissue Organization on Glucose Utilization of the Retina in Rat Serum*

Tissue Organization	No. of Experiments	Glucose per Retina, Mg.
Intact retina	55	0.488 ± 0.013
Fragmentation of retina by scissors	4	0.42
Homogenization of retina	6	0

TABLE 2.—*Influence of Various Media on Glucose Utilization of the Retina*

Medium	No. of Experiments	Glucose, per Retina, Mg.
Rat serum	55	0.488 ± 0.013
Human serum	6	0.42
Krebs-Ringer phosphate solution	101	0.214 ± 0.006
Krebs-Ringer bicarbonate solution	6	0.47
Saline serum substitute, bicarbonate buffer *	22	0.43 ± 0.02
Saline serum substitute, bicarbonate buffer, without organic substrate (fumarate, glutamate, pyruvate)	10	0.417 ± 0.033
Saline serum substitute, bicarbonate buffer, without pyruvate	7	0.49 ± 0.034

* The composition of the saline serum substitute is as follows: [11]

80 pt. 0.9% NaCl (0.154 M)
4 pt. 1.15% KCl (0.154)
3 pt. 0.11 M $CaCl_2$
1 pt. 2.11% K H_2PO_4 (0.154 M)
1 pt. 3.82% $MgSO_4 \cdot 7H_2O$
21 pt. 1.3% $NaHCO_3$ (0.154 M), treated with CO_2 until pH is 7.4
4 pt. 0.16 M Na-pyruvate
7 pt. 0.1 M Na-fumarate
4 pt. 0.16 M Na-L-glutamate
5 pt. 0.3 M (5.4%) glucose
} Prepared by neutralizing a solution of the acids with M $NaHCO_3$ solution

PRESENT INVESTIGATION

Method.—Normal white rats, weighing 90 to 120 gm., were used in all experiments. The rats were anesthetized with a 1% solution of pentobarbital sodium (1 cc. per 100 gm. of body weight) and were bled by cutting the aorta and inferior vena cava. The eyes were enucleated and placed in an isotonic saline solution (6.582 gm. NaCl, 0.183 gm. $MgCl_2$, and 0.272 gm. $CaCl_2$ in 1,000 cc. H_2O). The globe was cut around just posterior to the ora serrata and the retina removed in one piece from the underlying choroid. The two retinas from the same animal were suspended in a 10 ml. Erlenmeyer flask containing 1 ml. of the desired medium. The glucose concentration in each medium was 0.2% unless otherwise stated. The flask was aerated with

7. Oguchi, T.: Experimentelle Studien über den Einfluss der verschiedenartigen Bedingungen auf den Stoffwechsel der Netzhaut: I. Der physiologische Stoffwechsel der Kaninchennetzhaut in Ringerlösung und in Serum, Acta Soc. ophth. Japan **40**:1568, 1936.

8. Shaffer, M.; Chang, T. H., and Gerard, R. W.: Influence of Blood Constituents on Oxygen Consumption in Nerve, Am. J. Physiol. **111**:697, 1935.

oxygen for 30 seconds, stopped with a rubber stopper, and incubated for 2½ hours in a water bath at 37 C., with constant shaking in an automatic rocker. The amount of glucose in the medium was determined according to the method of Somogyi before and after incubation and the glucose utilization calculated therefrom. Results were expressed per retina, since the weight of the retinas used was almost constant. For three groups of 16 retinas each the respective weights and their standard deviations were as follows: 1.62 mg. ± 0.21 (13%); 1.65 mg. ± 0.17 (10.3%); 1.54 mg. ± 0.19 (12.3%). For weighing, the retinas were removed from the flask at the end of the experiment, rinsed, and dried at a temperature of 105 to 110 C.

Results.—The results are presented in Tables 1, 2, 3, and 4.

TABLE 3.—*Addition of Organic Substrate to Krebs-Ringer Phosphate Solution*

Krebs-Ringer Phosphate Solution (KRP)	Organic Substrate	No. of Experiments	Glucose per Retina, Mg.
KRP	.....	101	0.214 ± 0.006
KRP	0.005 M pyruvate; 0.005 M fumarate, 0.005 M glutamate.....	6	0.715 ± 0.05
KRP	0.005 M glutamate + 0.005 M fumarate.....	5	0.601 ± 0.029
KRP	0.0005 M glutamate + 0.0005 M fumarate.....	4	0.350 ± 0.06
KRP	0.005 glutamate + 0.005 M pyruvate.....	12	0.300 ± 0.006
KRP	0.005 M fumarate + 0.005 M pyruvate.....	11	0.380 ± 0.029
KRP	0.01 M glutamate.....	4	0.500 ± 0.017
KRP	0.005 M glutamate.....	13	0.412 ± 0.02
KRP	0.001 M glutamate.....	4	0.365 ± 0.006
KRP	0.01 M fumarate.....	6	0.465 ± 0.011
KRP	0.005 M fumarate.....	8	0.43 ± 0.024
KRP	0.001 M fumarate.....	3	0.350 ± 0.048
KRP	0.0005 M fumarate.....	2	0.318 ± 0.006
KRP	0.00025 M fumarate.....	2	0.319 ± 0.096
KRP	0.005 M pyruvate.....	6	0.284 ± 0.016

TABLE 4.—*Influence of Various Ions on the Glucose Utilization of the Retina*

Medium	No. of Experiments	Glucose per Retina, Mg.
Saline serum substitute, bicarbonate buffer.....	22	0.43 ± 0.02
Saline serum substitute, phosphate buffer without Ca and low in bicarbonate and CO_2 *.....	10	0.712 ± 0.04
Saline serum substitute, phosphate buffer, without Ca and low in bicarbonate and CO_2, without pyruvate, fumarate, or glutamate.....	9	0.410 ± 0.032
Saline serum substitute, low in phosphate, bicarbonate, and CO_2 †.....	13	0.486 ± 0.05
Saline serum substitute, low in phosphate, bicarbonate, and CO_2, without pyruvate, fumarate, or glutamate.....	7	0.189 ± 0.026
Saline serum substitute, bicarbonate, buffer without K (Na instead of K)	5	0.395 ± 0.031
Saline serum substitute, bicarbonate, buffer without Na (K instead of Na)	7	0.510 ± 0.008

* 83 pt. 0.9% NaCl
4 pt. 1.15% KCl
1 pt. 2.11% KH_2PO
1 pt. 3.82% $MgSO_4 \cdot 7H_2O$
3 pt. 1.3% $NaHCO_3$
18 pt. Na-phosphate buffer (160 pt. 0.1 M Na_2HPO_4 and 25 pt. 0.1 M NaH_2PO_4)
4 pt. 0.16 M Na-pyruvate
7 pt. 0.1 M Na-fumarate
4 pt. 0.16 M Na-L-glutamate
5 pt. 0.3 M (5.4%) glucose

† 85 pt. 0.9% NaCl
4 pt. 1.15% KCl
3 pt. 0.11 M $CaCl_2$
1 pt. 2.11% KH_2PO_4
1 pt. 3.82% $MgSO_4 \cdot 7H_2O$
3 pt. 1.3% $NaHCO_3$
3 pt. Na-phosphate buffer
4 pt. 0.16 M Na-pyruvate
7 pt. 0.1 M Na-fumarate
4 pt. 0.16 M Na-L-glutamate
5 pt. 0.3 M (5.4%) glucose

COMMENT

The glucose utilization of the retina is apparently dependent on the integrity of the cells. When the retina was cut by scissors into 20 pieces or more, the utilization of glucose was not appreciably different from that found with intact retina (four experiments—glucose utilization, 0.42 mg. glucose per retina). On

the other hand, when the tissue was ground up in a tissue grinder, the glucose utilization fell to zero (six experiments—glucose utilization, zero; Table 1).

There was an increase of 127% in the glucose utilization in rat-serum medium over that found in Krebs-Ringer phosphate. No difference was noted between autologous and homologous serum, and there was only a slight decrease when heterologous (human) serum was substituted as a medium.

In bicarbonate buffer the glucose utilization of the retina reached approximately the level of that in rat serum (Table 2). This is in complete agreement with the results obtained in measurements of the respiratory rate. Bicarbonate seems to be necessary for the synthesis of the Krebs-cycle intermediates. This was indicated by the observations of Crane and Ball [9] that the retina was highly active for CO_2 fixation and by our present findings concerning the relationship of the optimal glucose uptake to bicarbonate and Krebs-cycle members or their precursors. If bicarbonate was absent from the medium or present in a low concentration, the organic substances (pyruvate, fumarate, and glutamate) were able to replace the bicarbonate (Tables 3 and 4). In the presence of bicarbonate in a physiological concentration the removal of the organic substances from the medium had no effect whatever on the glucose utilization of the retina (Table 2). The addition of all three organic substances (pyruvate, fumarate, and glutamate) to Krebs-Ringer phosphate solution provided the best conditions for glucose utilization of the retina (Table 3). Removal of pyruvate from the serum-saline substitute with bicarbonate buffer increased somewhat the glucose utilization (Table 2). Pyruvate seemed to act as a competitor of glucose, and its presence decreased the glucose utilization. Dickens [10] and co-workers noted that the respiration of brain was considerably raised when calcium and magnesium were left out of the usual medium. With the other tissues tested (kidney, testis, tumor) the respiration remained unaffected. According to Krebs,[11] the Q_{O2} values for brain cortex in phosphate-buffered medium low in bicarbonate and CO_2 without calcium was about 37%—87% higher than in medium low in phosphate and bicarbonate containing calcium. In the case of liver the differences were smaller. In retina the glucose utilization increased by 47% in the phosphate-buffered medium low in bicarbonate and CO_2 without calcium (Table 4) over the glucose utilization obtained in the medium low in phosphate and bicarbonate containing calcium.

Serum substitute without potassium caused a slight decrease in the glucose utilization, but when potassium was substituted for sodium there occurred a noticeable rise in the glucose utilization (Table 4). Dickens [10] and co-workers reported that a high concentration of potassium accelerated the brain respiration, and they considered this finding a result of a physical change in the tissue. Robbie and Leinfelder [12] noted that the respiration of brain slices was increased on addition

9. Crane, R. K., and Ball, E. G.: Factors Affecting the Fixation of $C^{14}O_2$ by Animal Tissues, J. Biol. Chem. **188**:819, 1951; Relationship of $C^{14}O_2$ Fixation to Carbohydrate Metabolism in Retina, ibid. **189**:269, 1951.

10. Dickens, F., and Greville, G. D.: Metabolism of Normal and Tumor Tissue: XIII. Neutral Salt Effects, Biochem. J. **29**:1468, 1935.

11. Krebs, H. A.: Body Size and Tissue Respiration, Biochim. et biophys. acta **4**:249, 1950.

12. Robbie, W. A., and Leinfelder, P. J.: Use of Isolated Retinal Tissue in Studies of the Metabolism of the Central Nervous Tissue, Am. J. Ophth. (No. 6, Pt. 2) **32**:208, 1948.

of 0.1 M solution of Kcl to a phosphate-buffered saline solution. The retina, on the other hand, showed, if anything, a slight decrease in the oxygen consumption in a medium containing potassium.[12] In our experiments potassium seemed to increase the glucose utilization of the retina.

SUMMARY

The influence of various media on glucose utilization of the retina was studied. The glucose utilization was dependent on the integrity of the cells, since trituration of the retina in a tissue grinder abolished entirely its glucose utilization.

Serum was a much better medium for glucose utilization of the retina than was Krebs-Ringer phosphate solution. The results with Krebs-Ringer bicarbonate solution, on the other hand, equaled those obtained with serum.

Addition of pyruvate, fumarate, and glutamate to Krebs-Ringer phosphate solution enhanced appreciably the glucose utilization of the retina, which in this medium reached its highest value. These organic substances could replace bicarbonate in any medium without lowering the glucose utilization of the retina.

Removal of the calcium from the serum substitute of Krebs [11] increased the glucose utilization of the retina. The same effect was gained if potassium was substituted for sodium in the above-mentioned medium.

VARIATION OF CRITICAL FLICKER FREQUENCY IN THE NASAL VISUAL FIELD

Relation to Variation in Size of the Entrance Pupil and to Stray Light Within the Eye

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THE LIMINAL rate of alternation of a series of spatially superimposed flashes of light which is just seen to flicker is known as the critical flicker frequency. Recently attempts have been made to use this psychophysical measurement in a variety of clinical situations to detect, among other things, early fluctuations in body function, including acid-base changes,[1] cardiovascular changes,[2] hypoxia,[3] intelligence scores,[4] neurological disorders,[5] and "fatigue."[6] Ophthalmologically, there have been attempts to measure the critical flicker frequency in different regions of the visual field in lieu of, or in addition to, the more traditional visual field measurements. Particularly active in such work have been Phillips,[7] Riddell,[8] Hylkema,[9]

From the College of Optometry, Pacific University.

1. Hendley, C. D., and Alpern, M.: Effect of Acid-Base Changes on Flicker Fusion Frequency, abstracted, Am. J. Physiol. **163**:721, 1950. Alpern, M., and Hendley, C. D.: Visual Functions as Indices of Physiological Changes in the Acid-Base Balance of the Blood, Am. J. Optom. **29**:301, 1952.

2. Krasno, L. R., and Ivy, A. C.: Response of the Flicker Fusion Threshold to Nitroglycerine and Its Potential Value in the Diagnosis, Prognosis, and Therapy of Subclinical and Clinical Cardiovascular Disease, Circulation **1**:1267, 1950.

3. Seitz, C. P.: Effects of Anoxia on Visual Functions: A Study of Critical Frequency, Arch. Psychol. **36**:257, 1940.

4. Halstead, W. C.: Brain and Intelligence: A Quantitative Study of the Frontal Lobes, Chicago, University of Chicago Press, 1947, p. 68. Tanner, W. P.: A Preliminary Investigation of the Relationship Between Visual Fusion of Intermittent Light and Intelligence, Science **112**: 201, 1950.

5. Battersby, W. S.; Bender, M. B., and Teuber, H. L.: Effects of Total Light Flux on Critical Flicker Frequency After Frontal Lobe Lesions, J. Exper. Psychol. **42**:135, 1951.

6. Simonson, E., and Enzer, N.: Measurement of Fusion Frequency of Flicker as a Test for Fatigue of the Central Nervous System: Observations on Laboratory Technicians and Office Workers, J. Indust. Hyg. & Toxicol. **23**:83, 1941. Brozek, J., and Keys, A.: Flicker Fusion Frequency as a Test of Fatigue, ibid. **26**:169, 1944.

7. Phillips, G.: Perception of Flicker in Lesions of the Visual Pathways, Brain **56**:464, 1933.

8. Riddell, L. A.: Use of Flicker Phenomenon in the Investigation of the Field of Vision, Brit. J. Ophth. **20**:385, 1936.

9. Hylkema, B. S.: Examination of the Visual Field by Determining Fusion Frequency, Acta ophth. **20**:181, 1942.

Weekers and Roussel,[10] and, in this country, Mayer and Sherman [11] and Miles.[12] The approach of such investigators has been to set up a series of "norms" depending upon the apparatus and stimulus conditions and to consider any deviations from such values as clinically significant early visual field changes.

Concomitant with the developments in the clinical study of flicker, a number of laboratory investigations have been carried out under a wide variety of stimulus situations to study the variation of the critical flicker frequency in various parts of the visual field.[13]

In spite of the voluminous amount of work on this problem, there is still no clear-cut answer to the simple question whether the critical flicker frequency for the center of the visual field is higher than, equal to, or lower than that for the peripheral field. In fact, all three answers have been given at one time or another, and there seems to be no obvious way of ordering the discrepancies in the data. For example, it has been suggested by some investigators that the size of the stimulus area could account

10. Weekers, R., and Roussel, F.: La mesure de la fréquence de fusion en clinique, Docum. ophth. **2**:130, 1948.

11. Mayer, L. I., and Sherman, I. C.: A Method of Flicker Perimetry, Am. J. Ophth. **21**: 390, 1938.

12. Miles, P. W.: (*a*) Flicker Fusion Frequency in Amblyopia ex Anopsia, Am. J. Ophth. **32**:225, 1949; (*b*) Flicker Fusion Fields: I. Effect of Age and Pupil Size, ibid. **33**:769, 1950; (*c*) Flicker Fusion Fields: II. Technique and Interpretation, ibid. **33**:1069, 1950; Flicker Fusion Fields: IV. Neuro-Ophthalmic Lesions, ibid. **34**:51, (No. 5, Pt. 2), 1951.

13. (*a*) Lythgoe, R. J., and Tansley, K.: Adaptation of the Eye: Its Relation to the Critical Flicker Frequency, Medical Research Council, Special Report Series, No. 134, London, His Majesty's Stationery Office, 1929. (*b*) Granit, R.: Comparative Studies on the Peripheral and Central Retina: I. On Interaction Between Distant Areas in the Human Eye, Am. J. Physiol. **94**:41, 1930. (*c*) Granit, R., and Harper, P.: Comparative Studies on Peripheral and Central Retina: II. Synaptic Reactions in the Eye, ibid. **95**:211, 1931. (*d*) Granit, R., and Von Ammon, W.: Comparative Studies on Peripheral and Central Retina: III. Some Aspects of Local Adaptation, ibid. **95**:229, 1931. (*e*) Creed, R. S., and Ruch, T. C.: Regional Variations in Sensitivity to Flicker, J. Physiol. **74**:407, 1932. (*f*) Ross, R. T.: The Fusion Frequency in Different Areas of the Visual Field: I. The Foveal Fusion Frequency, J. Gen. Psychol. **15**:133, 1936; (*g*) The Fusion Frequency in Different Areas of the Visual Field: II. The Regional Gradient of Fusion Frequency, ibid. **15**:161, 1936; (*h*) The Fusion Frequency in Different Areas of the Visual Fields: III. Foveal Fusion Frequency and the Light-Dark Ratio for Constant Retinal Illumination of Fusion, ibid. **18**:111, 1938. (*i*) Hecht, S., and Verrijp, C. D.: Influence of Intensity, Color and Retinal Location on the Fusion Frequency of Intermittent Illumination, Proc. Nat. Acad. Sc. **19**:522, 1933. (*j*) Hecht, S.; Shlaer, S., and Verrijp, C. D.: Intermittent Stimulation by Light: II. Measurement of Critical Fusion Frequency for the Human Eye, J. Gen. Physiol. **17**:237, 1933. (*k*) Hecht, S., and Verrijp, C. D.: Intermittent Stimulation by Light: III. The Relation Between Intensity and Critical Fusion Frequency for Different Retinal Locations, ibid. **17**:251, 1933; (*l*) IV. A Theoretical Interpretation of the Quantitative Data of Flicker, ibid. **17**:269, 1933. (*m*) Brooke, R. T.: The Variation of Critical Fusion Frequency with Brightness at Various Retinal Locations, J. Optic. Soc. America **41**:1010, 1951. (*n*) Lloyd, V. V.: A Comparison of Critical Fusion Frequencies for Different Areas in the Fovea and Periphery, Am. J. Psychol. **65**:346, 1952. (*o*) Monnier, M., and Babel, J.: La fréquence de fusion de la rétine chez l'homme: Variations du seuil subjectif et du seuil électro-rétinographique selon le territoire rétinien stimulé, Helvet. physiol. et pharmacol. acta **10**:42, 1952. (*p*) Best, W.: Die Abhängigkeit der Flimmerfrequenz von der Reizflächengrösse und dem Ort der gereizten Netzhautstelle unter besonderer Berücksichtigung der Schwellenreizlichtstärke, von Graefes Arch. Ophth. **152**:99, 1951.

for the discrepancy, since measurements with a small (12-minute) stimulus showed the critical flicker frequency of the central field to be higher than that of the peripheral field,[13e] and since Granit and Harper [13c] found that for small test objects the critical flicker frequency was higher centrally than peripherally, whereas for large test objects the reverse was true. On the other hand, using a test object 2 degrees in diameter, Granit and Von Ammon [13d] found the critical flicker frequency to be much higher peripherally than centrally; Lloyd [13n] found (at very high luminance levels of the flickering light) that the critical flicker frequency was about the same centrally as at 20 degrees inferiorly, while Hecht and Verrijp [13i-l] and Brooke [13m] found the critical flicker frequency appreciably higher centrally than peripherally.

These discrepancies have been explained by the assumption that with a continuously luminous surround the critical flicker frequency would be lower peripherally than centrally. Fry and Bartley,[14] Geldard,[15] and others [16] have shown that if, as was usually the case, the surround was equal to the Talbot luminance of the flickering light, the central critical flicker frequency was higher with than without the surround. Another explanation suggested that the variation was due to the fact that at medium luminance levels of the flickering light the critical flicker frequency centrally was higher than peripherally, but that at higher luminance levels this was no longer the case.[13n]

Some of the investigations previously reported have been carried out with certain of the known variables not rigidly controlled. Particularly prominent in this respect would be control of the pupil size, which, although long known to be a factor, has rarely been controlled. In addition, many previous investigators have made measurements at only large intervals in the visual field. In the present study it was attempted to control such conditions as rigidly as possible. The controls included pupil size, the absence of a surround, intense Talbot luminance of the flickering light, and measurements of critical flicker frequency at intervals of every 1 degree into the nasal visual field.

APPARATUS

The apparatus used in the present study is illustrated in Figure 1. The source (S) was a 32-candle-power automobile head lamp, which was connected to a 6-volt direct-current generator. A storage battery was connected in parallel with the generator in order to obviate small fluctuations in voltage. The source was housed in a lamp housing, containing also a convex lens (L_1), which focused the light from the source on a ground-glass screen (G), immediately in front of which was a 1 mm. aperture (A_1). A sectored disk (D) containing equal intervals of interruption (light-dark ratio equal to unity) was mounted on a variable-speed friction drive in such a position that it interrupted the light beam just before it focused on the ground-glass screen. This permitted a very sharp cut-off. The aperture (A_1) was mounted at one end of a 100-cm.-long brass tube (1⅜ in. [35 mm.] diameter), in the other end of which was mounted a convex lens of 50-cm. focal length (L_2). In front of this lens was placed a second aperture (A_2), the field stop of the optical system. The lens (L_2) formed an image of the aperture A_1 at a distance of 100 cm. in front of the lens, and the observer's eye was placed so that the center of his entrance pupil coincided with the center of this image. As assistance to the subject in maintaining this position

14. Fry, G. A., and Bartley, S. H.: Effect of Steady Stimulation of One Part of the Retina upon the Critical Flicker Frequency in Another, J. Exper. Psychol. **19**:351, 1936.

15. Geldard, F. A.: Flicker Relations Within the Fovea, J. Optic. Soc. America **24**:299, 1934.

16. Graham, C. H., and Granit, R.: Comparative Studies on the Peripheral and Central Retina: VI. Inhibition, Summation, and Synchronization of Impulses in the Retina, Am. J. Physiol. **98**:664, 1931.

rigidly fixed, his head was held fixed by a wax-biting board, which was attached to a biting board assembly,[17] fastened firmly to a table. This permitted accurate control of the amount of light entering the observer's eye, since the image formed in the center of his entrance pupil was always smaller than the smallest possible entrance-pupil diameter. In front of the aperture A_2 was placed a large opaque screen (M), which prevented stray light in the room from reaching the observer's eye. The brass tube and cardboard box which completely surrounded the lamp housing, except for a small round hole through which the focal beam passed, also assisted in this respect.

The observer's fixation was controlled by a small (1.5 volt) naked-filament light bulb (F), which was made completely opaque except for a 2-mm. opening facing the observer. This was mounted in a battery handle from an ophthalmoscope, which, in turn, was clamped to a track moving along in front of the screen (M). The luminance of this bulb was kept at the visibility threshold by variation of the rheostat on the battery handle, while the position of the fixation point could be varied horizontally by sliding the clamp along the track. The position of the fixation point could be determined from a scale mounted to the screen (M).

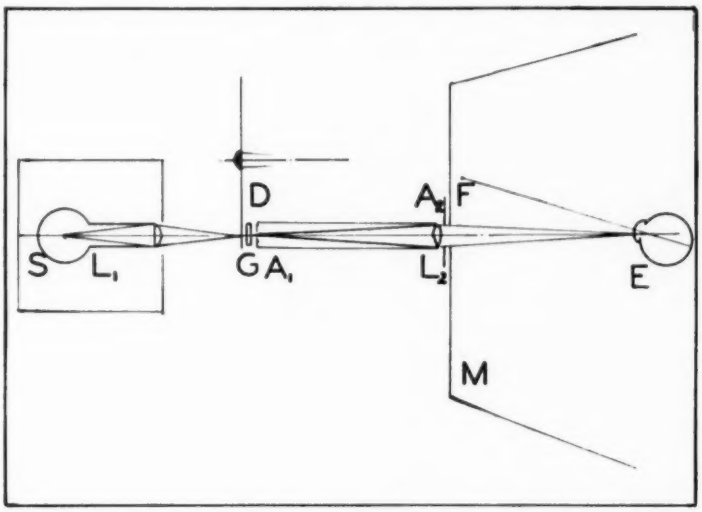

Fig. 1.—Apparatus used to measure the critical flicker frequency in various regions of the visual field.

The Talbot luminance of the flickering stimulus was determined by placing the entrance pupil of the Luckiesh-Taylor brightness meter in the plane of the image of the aperture A_1 formed by the lens L_2 while the disk was rotating at a speed above the critical flicker frequency. This luminance was found to be 1.28×10^4 foot-lamberts, which gave a retinal illuminance of 1.38×10^5 trolands.[17a] The test object subtended an angle of 0.9 degree at the center of the entrance pupil of the observer's eye.

PROCEDURE

Measurements were made on two observers using the psychophysical method of average error. Care was taken before each measurement to adapt the observer to the talbot luminance of the flickering stimulus. The observer reported when the flicker first disappeared as the experimenter

17. Drs. Merrill J. Allen and Glenn A. Fry, of the Ohio State University School of Optometry, loaned this assembly.

17a. The troland is a unit of retinal illuminance. One troland is the illuminance obtained when a surface having a luminance of 1 candle per quare meter is viewed through a pupil having an area of 1 sq. mm.

increased the speed of rotation of the disk. The speed of rotation of the disk was then decreased until the observer reported the first appearance of the flicker. The entire procedure was then repeated, bracketing the critical flicker frequency in this way, and for the second determination the speed of rotation of the disk was determined. This was done by counting, with a revolution counter and an electric timer, which could be read with an accuracy of 0.1 second, the number of rotations of the disk in 20 seconds. While the experimenter determined this value, the observer rested. The entire procedure was repeated five times and the mean of the five readings taken as the critical flicker frequency. The fixation point was then moved to a new position, and the critical flicker frequency for this point of the visual field was obtained in a like manner. Values for the critical flicker frequency were obtained in this way for the center of the visual

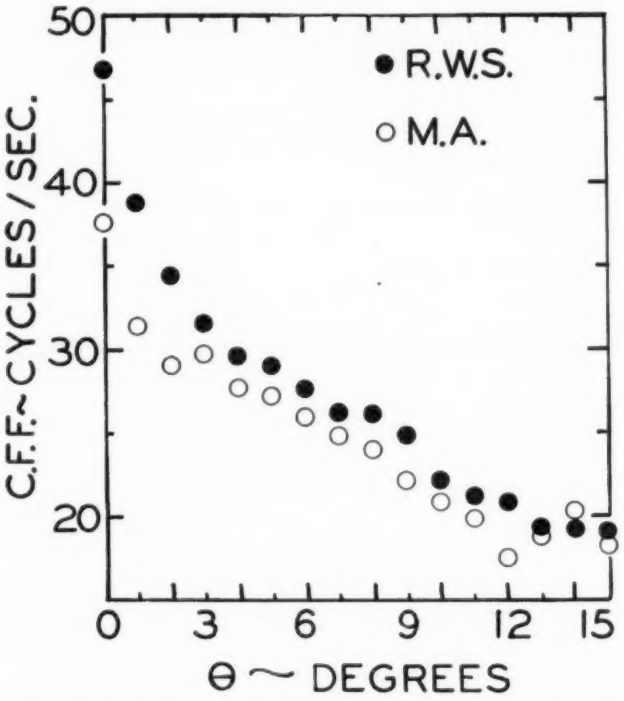

Fig. 2.—Variation of the critical flicker frequency with peripheral angle subtended by the distance from the fixation point to the center of the test field. Apparatus is as illustrated in Figure 1.

field and for each 1-degree interval along the horizontal meridian in the nasal visual field out to 15 degrees. The entire procedure was repeated on at least three separate days for each observer.

RESULTS

The results of this experiment are illustrated in Figure 2. In this figure the ordinates are values for the critical flicker frequency obtained by averaging the values obtained on different days. The abscissae are values for the angles subtended at the center of the entrance pupil by the distance from the center of the test field and the fixation point, expressed in degrees. The data for M. A. are given as open circles, and those for R. W. S., as filled circles. The two sets of data showed the same general

trends, although the data for R. W. S. showed usually a slightly higher critical flicker frequency than those for M. A. The data showed a fall in the critical flicker frequency as the test object was moved from the center of the visual field out along the horizontal meridian of the nasal field. This drop began as soon as the test object was withdrawn from the center of the field and continued out to the limit of the data. There was some tendency for the data to level off in the region of 15 degrees, although there is ample evidence that it would continue to fall farther out into the periphery.[13j-m]

The data in Figure 2 were in good agreement with those of Hecht and Verrijp [13l-l] and of Brooke,[13m] which were taken at much wider angle intervals. Since both these investigators used rigid controls in their experiments, it seemed likely that the discrepancy between these data and those found by other investigators might be due to the failure of the latter to prevent fluctuations in the size of the entrance pupil from affecting their results.

In order to test this possibility, the following experiments were performed. The lens (L_2) was replaced with a lens of sufficient focal length to render the image of the aperture (A_1) conjugate to the far point of the observer's eye.[18] The diameter of the aperture (A_1) in this case was increased to 2 mm., so that the visual angle subtended by the test object at the center of the observer's entrance pupil was equal to 0.11 degree. The data were obtained in a manner similar to that described for the data of Figure 2 except that beyond 3 degrees measurements were made only at every other degree. The results of this experiment are illustrated in Figure 3*A*, in which the ordinates and abscissae are the same as those of Figure 2.

The fundamental difference between this experiment and that illustrated in Figure 2 was that since the diameter (17.5 mm.) of the aperture A_2 was larger than the diameter of the largest possible entrance pupil, the aperture stop of the system had now become the pupil of the observer's eye instead of the aperture A_1, as was the case for the experiments in which the data of Figure 2 were obtained. This means that any fluctuation in the magnitude of the observers' photopupillary responses with variation in position in the field of the test object might have been a factor in the discrepancy between the two sets of data.

The data in Figure 3*A* show certain individual differences for the two observers. The data for M. A. (open circles) showed an immediate sharp increase in critical flicker frequency as the test object was moved from the center of the visual field out into the peripheral visual field. After reaching a maximum value at an eccentricity of 3 to 5 degrees, the critical flicker frequency began to fall and leveled off at a value somewhat higher than that for the central visual field. The data for R. W. S. (solid circles), on the other hand, showed a more gradual rise, which continued out to the highest eccentricity studied. In general, however, these results were in agreement with results reported by other investigators, who failed to control the variation of retinal illuminance related to variations in pupil size. There is evidence from such investigations that if the test target had been moved farther out into the peripheral field the critical flicker frequency would have begun to fall and eventually would have

18. In all the experiments reported, one of the observers (M. A.) wore the spectacle correction for his right eye (+ 3.37 sph. ⊃ + 1.37 cyl., axis 95), and the other observer (R. W. S.) did not wear the spectacle correction for his right eye (— 0.50 sph.). Consequently, the lenses (L_2) used for the two observers in this phase of the experimentation were different.

reached a value considerably lower than that found for the center of the visual field.[19] It was possible, though unlikely, that the discrepancy between the data of Figure 2 and those of Figure 3*A* might have been due to a difference in the apparent size or luminance of the test object in the two experiments. As a test of this possibility, the experiment illustrated in Figure 3*A* was repeated in an identical manner except that the observer's pupil was dilated by putting in 2 drops of mydriatic solution of 1% hydroxyamphetamine (Paredrine) hydrobromide and 5% eucatropine hydrochloride in the conjunctival sac of the observer, the second drop being instilled approximately 10 minutes after the first. After the mydriatic had taken

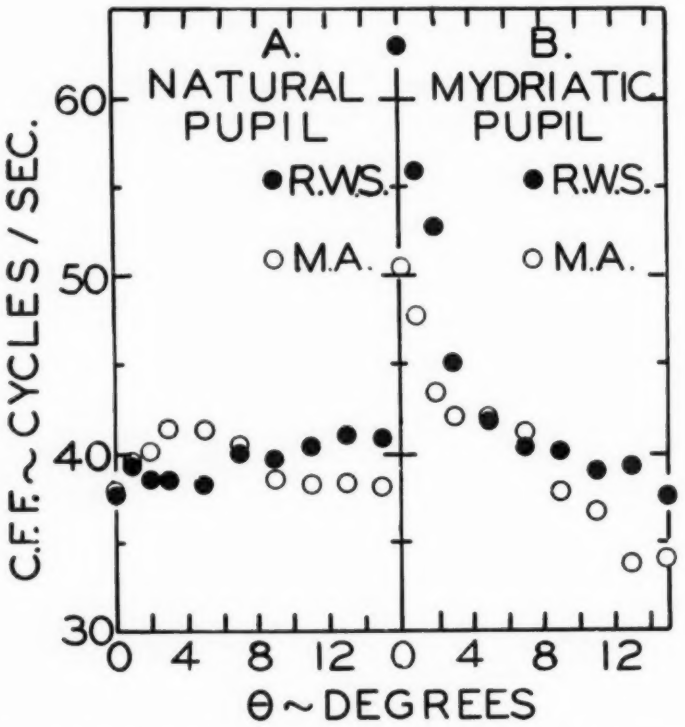

Fig. 3.—Variation of the critical flicker frequency with peripheral angle. Apparatus is as shown in Figure 1, except that the lens (L_3) has been replaced by a lens which renders A_1 conjugate to the far point of the observer's eye. *A* represents the observer's natural pupil; *B*, the observer's pupil dilated with mydriatic.

effect, as was evidenced by the complete absence of the photopupillary response in the eye concerned, the critical flicker frequency was measured in the various parts of the visual field.

The results of this experiment are plotted in Figure 3*B*, the ordinates and abscissae of which are the same as those of Figure 2 and Figure 3*A*. Since the characteristics of these data were essentially the same as those in Figure 2, with the

19. Phillips.[7] Riddell.[8] Hylkema.[9] Lythgoe and Tansley.[13a]

exception of the absolute values for the critical flicker frequency, the discrepancies between the regional variation in critical flicker frequency illustrated in Figures 2 and 3*B* and that illustrated in Figure 3*A* must have been associated with the influence of regional variations in the magnitude of the photopupillary response.

As a demonstration of the manner in which fluctuation in the size of the entrance pupil could enter into this problem, the following experiment was performed: The apparatus was essentially the same as that with which the data of Figure 3 were obtained except that a third aperture (A_3) was mounted immediately in front of the observer's cornea and accurately centered on his line of sight. This aperture was an iris diaphragm, the diameter of which could be varied. As in all previous measure-

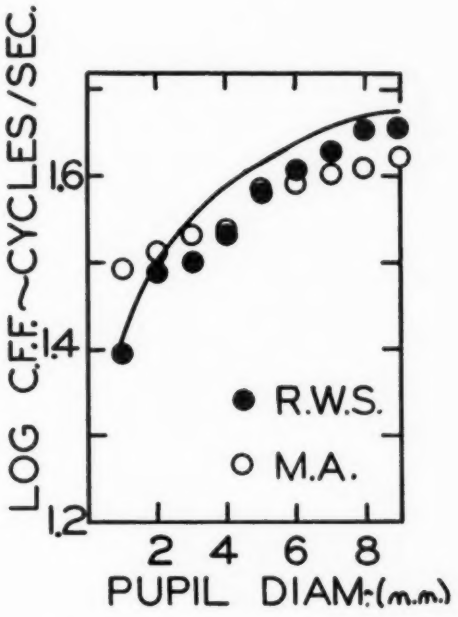

Fig. 4.—Variation in central critical flicker frequency with variation in the diameter of artificial pupil before the observer's eye.

ments, the observer was carefully adapted to the Talbot luminance of the flickering stimulus prior to the beginning of the determination. In order that the objection of Miles [12b] might be met, namely, that fluctuation in critical flicker frequency with variation of pupil size is due merely to inadequate adaptation to the low level of the stimulus with small artificial pupil diameters, one observer (M. A.) was dark-adapted for 30 minutes prior to the measurements and then adapted to the Talbot level of the stimulus for 5 minutes; each determination (i. e., 10 critical flicker frequency measurements) were then made while the observer was continuously fixating the test object. Measurements were made with increasing diameters of the artificial pupil with the exception of the smallest diameter, with which measurements were made at the conclusion of the experiment. All measurements were made with

the test object in the center of the visual field and with the observer's own pupil fully dilated with the same mydriatic and in exactly the same way as that described above.

The results of this experiment are plotted in Figure 4, in which the ordinates are the logarithms of the critical flicker frequency values, in cycles per second, and the abscissa values are diameters of the artificial pupil, in millimeters. In contradistinction to all the other data reported above, the data in Figure 4 represent measurements on one day only for each observer; those for M. A. (open circles) are the mean of 10 individual flicker determinations at each artificial pupil diameter, while those for R. W. S. (solid circles) are the mean of 5 separate flicker measurements.

In order to obtain some idea as to how the critical flicker frequency might vary with pupil size in association with the variation in the amount of retinal illuminance, we performed the following computation: The means of the data for central fixation for Brooke's [13m] two observers were plotted on a graph showing the logarithm of critical flicker frequency as ordinates and the logarithm of retinal illuminance as abscissae. The mean of the data for R. W. S. and M. A. for an artificial pupil of 2 mm. in diameter (the size of the artificial pupil in Brooke's experiment) was then used to estimate the retinal illuminance from this curve. With this value the luminance of the test object could be estimated, and once this value was obtained, the logarithm of the critical flicker frequency for pupils of various sizes could be predicted from this curve on the assumption that the retinal illuminance, in trolands, was equal to the product of the area of the pupil, in square millimeters, and the luminance of the test object, in candles per square meter. The curve which showed the estimated variation in critical flicker frequency with variation in diameter of the artificial pupil based upon these assumptions was drawn as the solid line in Figure 4.

The data in Figure 4 showed a clear increase in critical flicker frequency as the size of the artificial pupil increased. These data were in sharp contrast to those of Miles,[12a] which in general increased as the diameter of the artificial pupil was increased but leveled off when the diameter of the artificial pupil reached 3 mm., after which time any further increase in pupil size was unassociated with any further increase in critical flicker frequency. This difference could most likely be explained by Miles's failure to control the size of his observer's natural pupil. Since his values for critical flicker frequency were extremely high, it seemed probable that when the size of the artificial pupil reached values greater than 3 mm. the observer's natural pupil, rather than the artificial pupil, became the aperture stop of the optical system. Evidence in support of this assumption was also found in the fact that Miles's data for the peripheral field showed a continuous rise in critical flicker frequency with pupil diameter, an increase which would be predicted on the basis of the above experiments if the natural pupil was indeed the aperture stop for larger values of artificial-pupil diameter in central fixation in his experiments.

The data in Figure 4 seem to imply that the fluctuation of central critical flicker frequency with pupil size is most easily explained on the basis of the variation in retinal illuminance, together with the variation in the relative visual effectiveness of light passing through different regions of the observer's natural pupil. In 1933 Stiles

and Crawford [20] demonstrated that light passing through different regions of an observer's pupil was not equally effective in producing a visual effect but that, all other things being equal, light passing through the edge of a pupil of 8 mm. diameter was about one-fifth as effective visually as was light passing through the center of the pupil. This phenomenon has been verified by a number of independent investigators,[21] so that its existence can no longer be subject to serious question. It was manifested in the data of Figure 4 by the fact that for large artificial-pupil diameters the values dropped clearly below the theoretical values predicted if the variation in critical flicker frequency with pupil size were only a function of the variation in retinal illuminance. The discrepancies between the data for the two observers shown in Figure 4 can probably be related to errors of measurement, although the possibility that these data demonstrated individual differences in the Stiles-Crawford effect should not be overlooked.

COMMENT

The results of these experiments help to explain the confusion found in the literature with reference to the regional variation in critical flicker frequency. It is clear that a large amount of variability in results can be attributed to the failure of many of the investigators to control the size of the pupil in their experiments. This is true, for example, in the experiments of Miles, Riddell, Phillips, Lythgoe and Tansley, Ross, Creed and Ruch, Granit, Granit and Von Ammon, Granit and Harper, Hylkema, Weekers and Roussel, and others. Some of these investigators found a fall in the critical flicker frequency in the peripheral field, and some found a rise. Several investigators found both a fall and a rise, the result depending upon the size of the stimulus area. For example, increasing the size of the test object was associated with a marked increase in the peripheral critical flicker frequency, but only a very slight, if any, increase in the central critical flicker frequency. Granit and Harper, and perhaps others, have interpreted these findings as evidence of peripheral spatial summation and the absence of such summation effects in the central retina. In the light of the present experiments, it is more reasonable to suspect that the failure for the center to show a comparable rise when the area of the test object was increased may be related to the increased photopupillary response produced centrally by the increased area, but not peripherally. Evidence that supported this method of ordering the phenomena was offered by the data of Hecht and Smith, who found an increase in the critical flicker frequency of from 42 to 58 cps when the diameter of the centrally fixed test object was increased from 0.3 to 19 degrees and the size of the observer's entrance pupil was controlled.[22] As verification of these conjectures, it was found that in the present experiments when the apparatus was arranged in the way with which the data in Figure 3*A* were obtained, the drop in the critical flicker frequency as the test object was moved out into the peripheral field could be duplicated merely by decreasing the diameter of the aperture (A_1) to 1 mm. That this was more closely related to the variation in the magnitude of the photopupillary

20. Stiles, W. S., and Crawford, B. H.: Luminous Efficiency of Rays Entering the Eye Pupil at Different Points, Proc. Roy. Soc., London, s.B **112**:428, 1933.

21. Moon, P., and Spencer, D. E.: On the Stiles-Crawford Effect, J. Optic. Soc. America **34**:319, 1944.

22. Hecht, S., and Smith, E. L.: Area and Critical Fusion Frequency of Flicker, J. Optic. Soc. America **25**:199, 1935; Intermittent Stimulation by Light: VI. Area and Relation Between Critical Frequency and Intensity, J. Gen. Physiol. **19**:979, 1936.

response than it was to a decrease in area of the test object per se was evidenced by the fact that when the area was increased the fall could still be duplicated if variations in the magnitude of the photopupillary response were prevented from affecting the amount of light reaching the retina, as in the experiments illustrated by the data in Figure 2.

The above hypothesis suggested, then, that the appearance of a rise in critical flicker frequency as the test object was moved out into the peripheral field when the observer's natural pupil was the aperture stop of the system and when its diameter was not controlled was related to regional variations in the magnitude of the photopupillary response. According to this hypothesis, the photic stimulus in the center of the visual field should have been much more active in producing a pupillomotor response than the same stimulus in the peripheral field. Consequently, a flickering light in the center of the field, by reducing the diameter of the observer's pupil, would have reduced the amount of retinal illuminance and correspondingly caused the critical flicker frequency for the center to have been low with respect to the periphery, where the flickering light did not produce a very marked photopupillary response and the retinal illuminance was higher. This theory is based on the idea that a light in the center of the visual field would be much more effective in producing a photopupillary response than one in the peripheral field, and the available evidence seems to indicate that this is indeed the case,[23] at least for the light-adapted eye.[24]

A study of the literature of regional variations of critical flicker frequency in the light of the above hypothesis seemed to bring considerable order to the confusion. It explained not only the variation obtained with increased area but also the variation with increased intensity. The only experiments which did not appear to be in complete accord with this hypothesis were those of Lloyd,[13n] who stated that he used a 2.5 mm. artificial pupil and found the critical flicker frequency for a 2 degree area about as high peripherally as centrally for the higher luminance values of the flickering light. The results might be explained by the assumption that with this extremely large artificial pupil at the higher levels of luminance and central fixation the observer's natural pupil, rather than the artificial pupil, became the aperture stop of the system. At any rate, it is evident that Lloyd's own explanation of the discrepancy, i. e., that the fall (found by Hecht and Verrijp) was due to the presence of a continuously luminous surround, cannot be applied to the present experiments, since these were performed with a dark surround and still demonstrated a continuous drop as the test target was gradually moved into the periphery.

Critical flicker frequency in many ways resembles visual acuity, particularly in that the relation of visual acuity to illuminance is quite similar to the relation of critical flicker frequency to illuminance. In addition, measurements of central critical flicker frequency and central visual acuity have been correlated and a correlation coefficient of +0.909 found.[25] The regional variation in visual acuity has been studied

23. Duke-Elder, W. S.: Text-Book of Ophthalmology, Vol. 1, St. Louis, C. V. Mosby Company, 1932, p. 553.

24. Fry, G. A., and Allen, M. J.: Effect of Flashes of Light on Night Visual Acuity, Wright Air Development Center, Air Research and Development Command, United States Air Force, Wright-Patterson Air Force Base, Dayton, Ohio, Technical Report, No. 52-10, Pt. 2, Nov., 1951.

25. Peckham, R. H., and Arner, W. J.: Visual Acuity, Contrast, and Flicker, as Measures of Retinal Sensitivity, J. Optic. Soc. America **42**:621, 1952.

at some length,[26] and it is known that as the test object is gradually moved out into the peripheral visual field the acuity decreases at a very rapid rate.[27] This decrease in visual acuity in the peripheral visual field has been related to the cone distribution in the peripheral retina.[26a] If, as has been suggested by Hecht and Verrijp, the critical flicker frequency at extremely high levels of illuminance is exclusively a cone activity, it might be supposed that the critical flicker frequency should fall off into the periphery in much the same way as visual acuity does. In order to test this speculation, we replotted the means of the data for the two observers in Figure 2 in terms of the per cent of central critical flicker frequency and compared them with the data for peripheral visual acuity of Feinberg [26a] and of Mandlebaum and Sloan,[26b] plotted in terms of per cent of central acuity. Such a comparison demonstrated that the decrease in critical flicker frequency with increase in peripheral angle was much slower and much less than was the corresponding decrease in visual acuity in the peripheral visual field. This implied that if the regional variation in critical flicker frequency was related to the spatial distribution of cone photoreceptors, the relationship was certainly a different one than that relating visual acuity to the distribution of cones in the peripheral retina. Recently evidence has been offered which suggests that the original speculation of Hecht and Verrijp may be subject to question,[13m,n] and the physiology of the decrease of the critical flicker frequency with increase in peripheral angle subtended by the distance from the test object to the point of regard is, consequently, far from clear.

A possible explanation for the decrease in critical flicker frequency with increase in peripheral angle might be related to stray light within the eye. For example, it might be supposed that flicker discrimination was essentially a function of the central retina and that a peripheral flickering source would be seen to flicker when the rate of alteration of the light was so slow that the critical flicker frequency for the illuminance of the veiling glare in the central retina (resulting from the stray light cast by the flickering source) would be reached. Such conjectures were qualitatively verified by the fact that the peripheral critical flicker frequency was lower than the central critical flicker frequency (when pupil size was controlled) and by the fact that if the peripheral flickering source was arranged so that it was confined to the limits of the blind spot a measurement of critical flicker frequency was readily made, and the variability of such measurements was of about the order of magnitude that was obtained when the flickering light was in functional parts of the visual field. As a test of this hypothesis, an attempt was made to compute the critical flicker frequency for various values of θ using the Stiles-Holladay [28] equation for the distribution of scattered light within the eye as a function of the peripheral glare angle, together with Brooke's data for the relation between critical flicker frequency and illuminance [13m] for a 2 degree, centrally fixed white test object.

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26. (*a*) Feinberg, R.: A Study of Some Aspects of Peripheral Visual Acuity, Am. J. Optom. **26**:, 1949. (*b*) Mandelbaum, J., and Sloan, L. L.: Peripheral Visual Acuity, with Special Reference to Scotopic Illumination, Am. J. Ophth. **30**:581, 1947.

27. Low, F. N.: Peripheral Visual Acuity, A. M. A. Arch. Ophth. **45**:80, 1951.

28. Fry, G. A., and Alpern, M.: Effect of Flashes of Light on Night Visual Acuity, Wright Air Development Center, Air Research and Development Command, United States Air Force, Wright-Patterson Air Force Base, Dayton, Ohio, Technical Report, No. 52-10, Pt. 1, Nov., 1951. Fry, G. A., and Alpern, M.: The Effect of a Peripheral Glare Source upon the Apparent Brightness of an Object, J. Optic. Soc. America **43**:189-195, 1953.

Such analysis predicted a drop in critical flicker frequency as a function of θ that was much more rapid than the data of Figure 2. This showed that if the peripheral critical flicker frequency was a function of the scattered light within the eye the relationship must be different from that suggested by the Stiles-Holladay equation. As further demonstration that the peripheral flicker perception must be something more than just the perception of stray light, the following experiment was performed. The apparatus was arranged as illustrated in Figure 1, with the fixation point so arranged that the flickering field was completely contained within the blind spot. Thirty measurements of the critical flicker frequency were then made under the usual testing procedure. The fixation point was then moved so that the flickering light was located in the visible part of the field immediately adjacent to the blind spot temporally, with the nasal border of the flickering light touching the

TABLE 1.—*Critical Flicker Frequency at the Blind Spot and in Its Immediate Adjacent Neighborhood* *

Locus of Test Object	Mean Critical Flicker Frequency, Cycles/Sec.	Standard Deviation of Measurements	Standard Error of Mean
A Blind spot (16°)	7.37	± 1.10	± 0.21
B Beyond temporal blind spot border (20°)	18.38	± 0.89	± 0.51
C Beyond nasal blind spot border (12°)	19.30	± 1.67	± 0.96

* Number of determinations was 30.

TABLE 2.—*Statistical Analysis of Data in Table 1*

Rows of Table 1 Under Comparison	t	P
A and B	41.5	0.001
A and C	32.5	0.001
B and C	2.6	0.02

temporal border of the blind spot. After 30 measurements of the critical flicker frequency were made under these conditions, the fixation point was moved so that the temporal border of the flickering light touched the nasal border of the blind spot, and 30 measurements were made under these conditions. All measurements were made on one observer (R. W. S.) in one evening. The results were tabulated in Table 1. It is obvious from the data that when the test object was in the blind spot the critical flicker frequency was much lower than when the test object was on either side of the blind spot. The t test of significance was applied in order to determine whether or not these differences were statistically significant. The results of such an analysis were tabulated in Table 2. In this Table the first column gives the testing situations which were compared, as indicated by the letters signifying the rows in Table 1 which applied; the second column, the calculated t value, and the third column, the probability that such a difference between the two means in question could have occurred by chance. It is seen that all the differences were highly significant. It is therefore apparent that the critical flicker frequency presumably from the scattered light within the eye was considerably lower than critical

flicker frequencies of the functional parts of the visual field, indicating that factors other than stray light within the eye were closely related to the regional variation in critical flicker frequency found in these experiments.

SUMMARY

Measurements of the critical flicker frequency in the center and along the horizontal meridian in the nasal part of the visual field made every 1 degree for two observers showed that if variations in the size of the entrance pupil were prevented from influencing the amount of retinal illuminance the critical flicker frequency was successively less with increase in peripheral angle. If variations in the size of the entrance pupil were permitted to influence the amount of retinal illuminance, on the other hand, the critical flicker frequency for the peripheral field was higher than that for the central visual field. The decrease of the critical flicker frequency with increase in peripheral angle was much less than the corresponding decrease in visual acuity and than the decrease predicted if the perception of peripheral flicker were merely a function of scattered light within the eye. The variation of central critical flicker frequency with size of entrance pupil could be predicted with reasonable accuracy if consideration was given to the variation of retinal illuminance and to the Stiles-Crawford effect. The critical flicker frequency for the blind spot was significantly lower than that for immediately adjacent functional portions of the visual field.

Theta chapter of Omega Epsilon Phi Fraternity furnished financial assistance; John Faris, Ph.D., gave technical help in the design of the apparatus, and Dr. James L. Sears cooperated in dilating the observers' pupils in these experiments.

SURGICAL MANAGEMENT OF RETINAL DETACHMENT WITH RETINAL TEARS IN RETROLENTAL FIBROPLASIA

Report of Two Cases

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ALTHOUGH retinal detachment is a well-known phase of the cicatricial stage of retrolental fibroplasia, there have been no reports in the literature mentioning tears associated with these detachments. The majority of observers have believed that retinal separation in this disease results from tension or traction on the retina by organizing and contracting angioblastic tissue in the vitreous and by vitreous bands.[1] The purpose of this paper is to describe two cases of retrolental fibroplasia in which retinal detachments associated with definite retinal tears occurred.

Opinion is divided as to when reversibility in the disease is still possible, but most authors feel that spontaneous regression after the appearance of extensive retinal detachments is unlikely.[2] Furthermore, "no effective prophylaxis or therapy for retrolental fibroplasia" has yet been developed,[3] and there are no reports in the literature describing successful surgical management of retinal detachments in cases of retrolental fibroplasia. In both cases to be described the retinal detachments were treated successfully by routine surgical procedures, and in both the detached retina was satisfactorily reattached and the tear sealed. After the surgical replacement of the retina, the retrolental process in the eye regressed.

In both cases under consideration the primary disease was either relatively stable or beginning to regress in the fellow eye. No claim is made or intended that retinal detachment surgery produced regressive changes in retrolental fibroplasia. The value of operation in these cases lay in the preservation of the integrity of the retina so that the process of regression of the primary disease could continue.

From the Department of Ophthalmology, University of Rochester School of Medicine and Dentistry, and the Strong Memorial Hospital.

1. (*a*) Heath, P.: Retrolental Fibroplasia as a Syndrome: Pathogenesis and Classifications, A. M. A. Arch. Ophth. **44**:245-247 (Aug.) 1950; (*b*) Pathology of the Retinopathy of Prematurity: Retrolental Fibroplasia, Am. J. Ophth. **34**:1249-1259 (Sept.) 1951. (*c*) Owens, W. C., and Owens, E. U.: Retrolental Fibroplasia in Premature Infants, ibid. **32**:1-21 (Jan.) 1949; (*d*) Retrolental Fibroplasia in Premature Infants, A. M. A. Arch. Ophth. **44**:479-480 (Sept.) 1950.

2. Heath.[1a,b] Reese, A. B., and Blodi, F. C.: Retrolental Fibroplasia, Fifth Francis I. Proctor Lecture, Am. J. Ophth. **34**:1-24 (Jan.) 1951.

3. Zachiarias, L.: Retrolental Fibroplasia: A Survey, Am. J. Ophth. **35**:1426-1454 (Oct.) 1952.

REPORT OF CASES

CASE 1.—M. S., a white female infant, was born Jan. 23, 1952, weighing 975 gm. The infant was stabilized initially in an air lock. She was transferred to an Isolette 24 hours later and maintained under high oxygen levels. Ophthalmoscopic examinations were started Feb. 12. A persistent vasculosa lentis and hazy vitreous and cornea were noted in both eyes. No definite retinal pathology was discovered until Feb. 21, when small round superficial hemorrhages were seen bilaterally. Within a few days significant neovascularization and peripheral edema of the retina could be demonstrated in both eyes. High oxygen levels were maintained from March 6 until March 26. During this period the fundus picture remained relatively stable. The infant was, therefore, weaned from the high oxygen levels and discharged. The weight at discharge was 2,330 gm.

After discharge, the vessels of both fundi became more tortuous and more dilated. The extreme peripheral retina of both eyes could be seen to be raised in white, ledge-like, edematous detachments with prominent neovascularization. The annular ledge-like detachments could be examined all around the extreme periphery except at the inferior portion, where visualization was very difficult. In the left eye a diffuse and extensive area of retinal edema with detachment was seen extending inward from the temporal periphery toward the macula, but not reaching it. The baby was readmitted to the hospital and again placed in a high oxygen concentration. In the ensuing month no new neovascularity appeared, and the process began to regress. The ledge-like peripheral rolls of the thickened retina began to subside, and the retinal vessels became less dilated. The edematous detachment in the temporal periphery of the left eye regressed almost completely, and both eyes seemed in much better condition. The child was discharged on May 5.

Nine days later routine ophthalmoscopic examination disclosed beginning retinal detachment in the temporal periphery of the left eye. There were several large superficial hemorrhages in the detaching retina. On May 20, the detachment was more pronounced and extensive, and a tear was seen in the retina at the extreme temporal periphery at 2:30 o'clock. The tear was wedge-shaped; four days later it appeared larger. The right fundus showed only regressive changes in the blood vessels, and the peripheral ledge of edematous retina was less prominent. Dr. John F. Gipner saw the patient in consultation. He confirmed the diagnosis and agreed that a surgical approach was indicated.

The infant was admitted to the hospital on May 24. The following day, with the patient under endotracheal ether anesthesia, the retinal tear in the left eye was sealed with partially penetrating Kronfeld cautery punctures, and a delimiting barrage of these cautery punctures was placed around the margins of the detached area. A small amount of subretinal fluid was drained through several penetrating cautery puncture holes made in the region of the retinal tear. The baby was hospitalized under modified postoperative retinal detachment regimen until June 5, when she was discharged. On discharge her weight was 3,850 gm.

Since discharge the eyes have run parallel courses. The peripheral ledge-like, edematous rolls of retina subsided completely. The vessels became normal in caliber. At the time of this report, both disks are large and pale and exhibit some dragging of the vessels inferiorly and straightening of the vessels superiorly. Both maculae appear normal on ophthalmoscopic examination. Although the infant is myopic (— 9.00 sph. — 1.50 cyl., ax 180 in each eye), she can see with either eye.

CASE 2.—J. M., a white female infant, was born Aug. 18, 1952, weighing 2,460 gm. She was brought to the office for routine ophthalmoscopic examination on Sept. 27, 1952, when she was 39 days old.

The globes were white. The pupils were small, round, regular, and reactive. The anterior chambers appeared shallow. The left fundus showed mild fulness of the veins and some peripheral edema of the retina. There was no neovascularization, but the edematous peripheral retina was elevated. The right fundus showed similar changes. The temporal periphery of the right fundus showed vitreous hemorrhage, retinal detachment, and a large retinal tear, situated approximately 7 to 9 mm. from the ora serrata at 10 o'clock. Two subsequent examinations, the last with

the patient under anesthesia, confirmed the presence of the retinal tear and showed some progression of the detachment. The diagnosis was retrolental fibroplasia with retinal detachment and retinal tear in the right eye.

The infant was admitted to the hospital on Oct. 4, and the following day the tear was sealed and the detached area delimited with Kronfeld partially penetrating electrocautery punctures. Several penetrating cautery punctures were made in the region of the tear, and a very small amount of subretinal fluid was drained through them. The baby was discharged from the hospital on Oct. 19.

Subsequent follow-up examinations have shown the retina of the right eye to be properly in place, the detached area flat, and the tear sealed. In both fundi the early evidence of retrolental fibroplasia has shown regression.

COMMENT

In both cases described above the retrolental changes regressed in one eye without a detachment or retinal tear developing. In the fellow eye, however, when the detachment with retinal tear developed, it showed a progressive tendency, with no indication of regression or scarring down. The recognized treatment for true detachment of the retina with demonstrable retinal tears is surgical; accordingly, both infants were subjected to surgery. In the next case of this type I plan to use conservative treatment as a control on these cases.

The courses of the process in these cases suggest a possible explanation for the occasional case of "unilateral" retrolental fibroplasia. One may speculate that, while regression of a mild or moderate form of retrolental fibroplasia occurred in one eye, a retinal detachment with retinal tear occurred in the other and, in the absence of surgical intervention, complete detachment, with the formation of a retrolental membrane in that eye, was the result.

It is of interest that, once the detachment and retinal tear were properly treated, the two eyes ran identical courses in regression or inactivation of the original pathologic process.

The history of these cases provides interesting data on the reaction of infant eyes to detachment surgery. The thinness of the sclera makes careful control of the cautery current essential. Careful localization of the tear is likewise most important. These eyes are so small that it would be easy to cauterize an excessive amount of retina in attempting to seal the tear. Direct ophthalmoscopic visualization of the tear during the procedure is of great assistance. In the cases described the tear was sealed and a delimiting barrage of cautery punctures was thrown around the border of the detachment to discourage any further spread.

Although the infants were kept on their backs, and off their faces, during the postoperative period of hospitalization and for a short time thereafter, they were raised normally during feeding to prevent aspiration and were burped in the usual fashion. Restraints were not employed, since these devices caused excessive restlessness. Finally, although both eyes were bandaged at first, it became apparent that after 7 to 10 days of this treatment the infant was more irritated than benefited by the bandage on the eye not operated on. Since much of the infant's day was spent in sleep, the bandage on this eye was eliminated early. In both cases atropine sulfate, 1% in ointment form, was instilled in the surgically treated eye every other day for two weeks.

SUMMARY

Two cases of retrolental fibroplasia with uniocular retinal detachment associated with retinal tear are presented. It is believed that these cases are the first to be described in which retinal tears occurred in the retinal detachments of retrolental fibroplasia, and the first of successful surgical management of any phase of retrolental fibroplasia.

A possible explanation for the occurrence of "unilateral" retrolental fibroplasia is suggested.

The case histories offer strong arguments for the early and long-continued careful ophthalmoscopic observations of all premature infants and infants of low birth weight, with the hope of discovering the pathologic process at a time when it can be checked by surgical means.

332 Park Ave.

CAT-SCRATCH DISEASE AND PARINAUD'S OCULOGLANDULAR SYNDROME

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THE ETIOLOGY of Parinaud's oculoglandular syndrome has always been an enigma. In some cases the syndrome has been associated with tularemia; in many with leptothricosis, and in a few, with syphilis, tuberculosis, or sporotrichosis. In a large number of cases, however, a thorough search for an inciting factor has been of no avail, and the origin of the disease is surrounded with much uncertainty. Numerous reports concerning cat-scratch disease have appeared in the recent medical literature. In many instances Parinaud's oculoglandular syndrome is probably a form of the same disease entity in which the etiologic agent has gained entrance into the conjunctiva rather than into the skin.

In 1889 Parinaud [1] described cases of oculoglandular fever starting as conjunctivitis with granulations, associated with enlargement of regional lymph nodes and a moderate fever, which persisted for weeks. He believed that this disease was contracted from animals. Gifford,[2] in 1898, gave it the name of Parinaud's conjunctivitis, and since that time similar conjunctivitis, with retrotarsal granulation, regional lymphadenitis, and fever, whatever its cause, has been called Parinaud's oculoglandular syndrome. Determination of the cause has been a fascinating study, more so than the disease itself, which is self-limited and usually of only moderate severity. The finding of a type of tularemia with this oculoglandular syndrome was of extreme interest to all who had searched for an etiologic factor, but the paucity of cases in which a diagnosis of tularemia could be confirmed led to a search for other causes.

Verhoeff [3] was certain that the Leptothrix was the etiologic agent. He [4] believed that his method of obtaining material for study had resulted in his finding this organism in more cases than had generally been reported. He suggested that if the gray areas (macrophage foci) of conjunctival granulations were selected for sectioning and stained by special methods, a higher percentage of Leptothrices would be

1. Parinaud, H.: Conjonctivite infectieuse transmise par les animaux, Ann. ocul. **101**:252-253, 1889.

2. Gifford, H.: Five Cases of Parinaud's Conjunctivitis, Am. J. Ophth. **15**:193-200, 1898.

3. Verhoeff, F. H.: Leptotrichosis Conjunctivae (Parinaud's Conjunctivitis), Without Glandular Involvement: Report of a Case, Arch. Ophth. **25**:1-6 (Jan.) 1941.

4. Verhoeff, F. H.: Improved Method of Staining Within Tissues, Leptotriches of Parinaud's Conjunctivitis and Gram-Positive Micro-Organisms, J. A. M. A. **115**:1546-1547 (Nov. 2) 1940.

found. Castelli and Molina[5] found an organism similar to Hemophilus influenzae (Koch-Weeks bacillus) which on conjunctival injection produced follicles and swelling in experimental animals similar to Parinaud's conjunctivitis. They called this organism Microbacterium conjunctividis. Parinaud believed that it was the etiologic agent and that the disease was a clinical entity and not a syndrome.

Similar conjunctival lesions of different origin undoubtedly occur. Maldonado Allende and Joisen,[6] in reporting a tuberculous infection of the conjunctiva, asked that it be differentiated from sporotrichosis, tularemia, and leptothricosis. Macnie[7] reported a case of oculoglandular disease due to lymphogranuloma venereum. Francis,[8] in 70 odd cases of tularemia, found either that the agglutination test was always positive or that cultures from inoculated guinea pigs yielded the organism. The serum of patients with Parinaud's conjunctivitis usually failed to agglutinate Pasteurella tularensis. In view of the specificity of the test, he concluded that Parinaud's oculoglandular syndrome and oculoglandular tularemia were not the same disease.

Martin,[9] in 1942, reported two cases of Parinaud's syndrome in persons who handled rabbits. Mescheriakova,[10] in 1943, described a case of oculoglandular tularemia characterized by conjunctival ulceration, febrile onset, and involvement of the regional lymph nodes. Canamares Mareno,[11] in 1943, reported four cases of the oculoglandular syndrome with diffuse infiltration of the conjunctiva without ulceration but with preauricular adenopathy in which sporotrichosis was found in pus aspirated from the preauricular lymph node. Gifford,[12] in an article on leptothricosis conjunctivae, pointed out that Parinaud's syndrome might be caused by Mycobacterium tuberculosis, Pas. tularensis, or Leptothrix. In the differential diagnosis of leptothricosis, Gifford emphasized evidence of small areas of focal necrosis, eosinophilia of 3 to 10% in the peripheral blood with moderate febrile and systemic reaction, elements of thread mold in sections of the lesion, and, at times, the presence of Leptothrix in smears.

Theodore,[13] in 1945, found a yeast-like organism in a case of Parinaud's oculoglandular syndrome and proposed the opinion that this was the responsible pathogen. Tularemia was again reported as the etiologic agent in a case reported by Krutova [14]

5. Castelli, A., and Molina, L.: La "congiuntivite di Parinaud" (studio monografico e contributo sperimentale alla conoscenza dell' eziologia), Ann. ottal. e clin. ocul. **68**:81-127 (Feb.) 1940.

6. Maldonado Allende, I., and Joisen, M.: Sobre un caso de primoinfección tuberculosa de la conjunctiva, Arch. oftal. Buenos Aires **15**:625-636 (Dec.) 1940.

7. Macnie, J. P.: Ocular Lymphogranuloma Venereum, Arch. Ophth. **25**:255-279 (Feb.) 1941.

8. Francis, E.: Oculoglandular Tularemia, Arch. Ophth. **28**:711-741 (Oct.) 1942.

9. Martin, W. O., Jr.: Parinaud's Oculoglandular Syndrome: Treatment with Sulfathiazole, Am. J. Ophth. **25**:1493-1495 (Dec.) 1942.

10. Mescheriakova, A. V.: Oculoglandular Tularemia, Vestnik oftal. **22**:45, 1943.

11. Canamares Mareno, S.: Sporotrichosis conjunctivitis, Arch. Soc. oftal. hispano-am. **3**:222-224 (Sept.-Oct.) 1943.

12. Gifford, S. R., and Day, A. A.: Leptotrichosis Conjunctivae: Further Report, Arch. Ophth. **31**:423-426 (May) 1944.

13. Theodore, F. H.: Parinaud's Oculoglandular Syndrome Due to a Yeastlike Organism, Arch. Ophth. **33**:471-475 (June) 1945.

14. Krutova, A. H.: Primary and Secondary Eye Lesions in Tularemia, Vestnik oftal. **25**:23-26, 1946.

in 1946. Siniscal [15] reported oculoglandular tularemia in three cases, the blood agglutination test being negative for 10 days but becoming positive 2 to 3 weeks after onset of the disease. The reaction to the intradermal test was positive by the fourth day. In these cases of Siniscal's the disease was contracted from domestic house cats, in one from the house cat's sneezing into the face of the patient. Paulson,[16] in 1946, cultured Leptothrix from the conjunctiva of a patient affected with this oculoglandular syndrome. Radnót,[17] in 1947, reported a Parinaud-like syndrome caused by a virus of fowl plague, not so-called, but probably, the Newcastle virus.

Thygeson,[18] in 1947, while reviewing the clinical signs of Parinaud's oculoglandular syndrome, pointed out that leptothricosis, tuberculosis, lymphogranuloma venereum, syphilis, and oculoglandular tularemia should be considered etiologic factors in this disease. Kant [19] reported an unusual case of leptothricosis of the conjunctiva in which the preauricular lymph node suppurated. Paufique and Etienne,[20] in 1949, in a study of the etiology of the conjunctivoglandular syndrome of Parinaud, like Theodore, found a yeast-like organism. This was of the Blastomyces group, and the reaction to the intradermal test was positive. Allen,[21] in 1951, in reviewing the literature on Parinaud's syndrome, said that the disease was a clinical syndrome with a variety of causative agents, including Leptothrix, Myco. tuberculosis, Past. tularensis, and virus.

In 1950 Pesmé and Marchand [22] reported a case of conjunctivitis with swelling of the preauricular lymph nodes and fever in a 6-year-old child who had been bitten and scratched by a cat. An intradermal test with an antigen of cat-scratch fever obtained from the pus of lymph nodes of patients from Debré's laboratory gave a positive reaction both in this child and in a patient with Parinaud's syndrome one year after they had recovered. Brault and Charbonneau,[23] in the same year, reported a case of Parinaud's conjunctivitis from contact with a cat. Leptothrix was found in the conjunctival granuloma and in scrapings from the cat's mouth. Henry,[24] in 1950, reported eight cases of oculoglandular conjunctivitis, in seven of which there was a history of contact with cats. She also, believing that the disease was trans-

15. Siniscal, A. A.: Oculoglandular Tularemia: Report of 3 Cases with Unusual Contacts, Am. J. Ophth. **29**:698-710 (June) 1946.

16. Paulson, H. O.: Parinaud's Conjunctivitis, Am. J. Ophth. **29**:1166-1167 (Sept.) 1946.

17. Radnót, M.: Maladie oculo-glandulaire jusqu'à présent inconnue, Ophthalmologica **113**:106-107 (Feb.) 1947.

18. Thygeson, P.: Clinical Signs of Diagnostic Importance in Conjunctivitis, J. A. M. A. **133**:437-441 (Feb. 15) 1947.

19. Kant, A.: Unusual Case of Leptotrichosis Conjunctivae, Am. J. Ophth. **31**:607-608 (May) 1948.

20. Paufique, L., and Etienne, R.: Contribution a l'étude de l'étiologie du syndrome conjunctivo-ganglionnaire de Parinaud, Ann. ocul. **182**:455-460 (June) 1949.

21. Allen, J. H.: Lids, Lacrimal Apparatus and Conjunctiva: Review of Recent Literature, A. M. A. Arch Ophth. **45**:100-119 (Jan.) 1951.

22. Pesmé and Marchand: Sur un nouveau type de conjonctivite infectieuse probablement transmise par un chat, J. méd. Bordeaux **127**:127-129, 1950.

23. Brault, J., and Charbonneau, R.: Syndrome oculo-glandulaire de Parinaud, Union méd. Canada **79**:584-586 (May) 1950.

24. Henry, M.: Oculoglandular Conjunctivitis Due to Leptothrix, J. Pediat. **37**:535-544, (Oct.) 1950.

mitted by cats, tried to correlate the Leptothrix of the conjunctiva with that of the cat's mouth. Lavergne and associates,[25] in 1951, reported a case of Parinaud's oculoglandular syndrome in which the course was prolonged and in which the diagnostic tests for tularemia, tuberculosis, lymphogranuloma venereum, and syphilis were negative but the intradermal skin test with cat-scratch antigen gave a positive reaction.

A report of the following cases of oculoglandular syndrome seen over the past few years is submitted at this time because in retrospect the condition seems likely to have been cat-scratch disease. They were studied for all the usual etiologic agents, but no definite diagnosis could be made, the results of all tests being essentially negative. When an antigen of cat-scratch fever became available, however, the patients were called back, and all of them gave strongly positive skin reactions against this antigen.

Fig. 1 (Case 2).—Parinaud's oculoglandular syndrome, showing granulation of the retrotarsal conjunctiva of the upper left lid, in February, 1948. A skin test for cat-scratch fever was positive on April, 1952.

REPORT OF CASES

CASE 1.—D. H., a boy aged 5 years, was first seen Feb. 16, 1952, with a swollen right eye. Examination revealed conjunctivitis with granulation and right preauricular and submaxillary lymphadenopathy. He had played with a cat for the previous six months, frequently holding it near his face. Agglutination tests against Past. tularensis and Brucella abortus were negative and smear and culture failed to reveal Leptothrix. An intradermal test with cat-scratch fever antigen gave a positive response on April 3.

CASE 2.—Miss M. A., aged 23, was first seen Feb. 17, 1948. She had a swollen left upper eyelid (Fig. 1) and swelling of the lymph nodes of the left preauricular area and the carotid triangle, of six weeks' duration. The patient had two cats, one of which had had distemper. Agglutination tests against Past. tularensis were negative, and smear and culture of conjunctival secretion revealed no pathogens. A biopsy of the conjunctival granulations showed a thin surface layer of stratified squamous epithelium, beneath which granulation tissue, almost solidly packed with plasma cells, was observed. There were no epithelioid cells, and necrosis was not present. A few large macrophages were seen, but special staining revealed no organ-

25. de Lavergne, V.; Thomas, C.; Cordier, J., and Algan, B.: La forme oculoganglionnaire de la lymphoréticulose bénigne d'inoculation, Bull. et mém. Soc. méd. hôp. Paris **67**:985-987 (June 29-July 6) 1951.

isms. By March 13, 1948, the granuloma had entirely cleared up, and the swelling of the nodes had disappeared. On April 9, 1952, an intradermal test with an antigen of cat-scratch fever gave a positive reaction.

Fig. 2 (Case 3).—Parinaud's oculoglandular syndrome showing prominent preauricular and cervical lymphadenopathy. Conjunctival granulations were present (not shown) in September, 1948. Skin test for cat-scratch fever was positive on April, 1952.

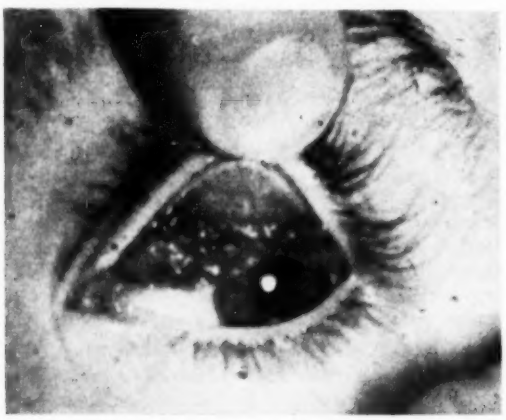

Fig. 3 (Case 4).—Parinaud's oculoglandular conjunctivitis, showing retrotarsal conjunctival granulations. Histologic examination revealed nonspecific granuloma.

Case 3.—E. K., a boy aged 11 years, was first seen Sept. 13, 1948, with a swollen left eye and preauricular and cervical lymphadenitis (Fig. 2). There was a systemic reaction, with a temperature of 101 F. A history of close contact with several cats was obtained. Agglutination tests against Past. tularensis were negative. Culture of conjunctival secretion yielded a growth of diphtheroid bacilli. These culture were studied by Dr. David Cogan, who could find no Leptothrix. After five days of symptomatic treatment in the hospital, the patient's condition

improved. An intradermal test with antigen of cat-scratch fever on April 9, 1952, gave a positive reaction.

CASE 4.—B. B., a boy aged 9 years, was seen Dec. 9, 1947, because of conjunctivitis with granulations in the right retrotarsal fold (Fig. 3) and preauricular and cervical lymphadenopathy. He had contact with cats only at his grandmother's home. Agglutination tests against Br. abortus and Past. tularensis were negative, and conjunctival cultures and smears revealed no pathogens. The Mantoux test gave a negative result in a dilution of 1:1,000. Biopsy of conjunctival granulation tissue showed a dense infiltrate of mononuclear cells, predominantly lymphocytes, with a few large macrophages. Necrosis and epithelioid cells were absent. The picture was that of a nonspecific granulomatous reaction. Approximately one month later recovery was almost complete. An intradermal test with an antigen of cat-scratch fever was positive on April 9, 1952.

COMMENT

When an antigen of cat-scratch disease became available for skin tests, these four patients, who had been seen over the previous several months, were tested with a view to establishing a relationship between Parinaud's oculoglandular syndrome and cat-scratch disease. The antigen was obtained from a fluctuant axillary lymph node of a patient with a typical history and findings of cat-scratch disease; it gave identical results with those obtained in parallel with a known antigen supplied by Dr. Worth B. Daniels in known cases of cutaneous cat-scratch disease. Numerous control tests gave negative results. The etiologic agent remains undetected, although most workers believe the agent is virus in nature.

Several factors seem significant in the search for the etiologic agent of Parinaud's syndrome. Transmission is usually not from patient to patient, as are most conjunctival diseases, and contact does not seem to be an important factor. Cases are usually sporadic, and usually only one member of a family is infected. Leptothrix has been held responsible for the disease oftener than any other organism; yet this organism has frequently been found as a saprophyte, unassociated with disease of any kind. Tularemia and yeast-like organisms have only in rare instances been proved etiologic. The fact that Parinaud's conjunctivitis is apparently not transmitted by human beings makes the likelihood of an animal vector seem reasonable. The clinical features, particularly the systemic reaction occurring in Parinaud's oculoglandular syndrome, are quite similar to those occurring in the cutaneous-glandular form of cat-scratch fever. The disease seems to vary from the latter entity only in its portal of entry and in the location of its "primary complex." Further correlation of positive skin tests against the antigen prepared from necrotic lymph nodes of the cutaneous-glandular disease would tend to indicate that the syndrome in the present cases has a common etiology with catch-scratch disease. Thus, Parinaud's oculoglandular syndrome is a manifestation of cat-scratch fever which differs in that the conjunctiva is the site of primary infection, the initial lesion being one of conjunctivitis with granulation, followed by regional lymphadenitis. Infections from cats by inhalation of infected material, transmitted by the nasopharyngeal or faucial route, has been reported by Daniels and MacMurray.[26] A cat's sneezing into the face resulted in the oculoglandular syndrome in Henry's case.

26. Daniels, W. B., and MacMurray, F. G.: Cat-Scratch Disease: Nonbacterial Regional Lymphadenitis, A. M. A. Arch. Int. Med. **88**:736-751 (Dec.) 1951.

Rubbing the eyes after handling a cat with infection of the conjunctiva is suggested as a possible means of transmission of this disease.

No new cases of oculoglandular syndrome have been observed since the antigen has become available. It has been well demonstrated by these cases and by the cases reported by others that the reaction to the skin test remains positive for a considerable period. How soon it will become positive in the oculoglandular syndrome remains to be determined; this factor becomes important in the early determination of the cause in a given case of the syndrome.

SUMMARY

Evidence gained from the study of four cases of Parinaud's oculoglandular syndrome indicates that the etiologic agent in these cases was most probably that of cat-scratch disease. Positive reactions to skin tests with cat-scratch antigen, a history of exposure to cats, similar pathogenesis, and similar constitutional symptoms are the main features of correlation.

NOTE:—Since this paper was submitted for publication, Daniels and MacMurray [27] have reported that Parinaud's oculoglandular syndrome can be caused by cat-scratch disease. They cite Paufique and Etienne [20] and Pesmé and Marchand [22] and also the work of Clément and associates [28] to substantiate the relation between cat-scratch disease and the oculoglandular syndrome. They also state that E. R. Peterson has encountered two such cases and has described one of them, in which a strongly positive intradermal reaction with the antigen of cat-scratch disease was obtained five years after the patient's recovery from Parinaud's oculoglandular syndrome.

921 Lincoln Way East (Dr. Cassady).

531 North Main Street (Dr. Culbertson).

27. Daniels, W. B., and MacMurray, F. G.: Cat Scratch Disease: Nonbacterial Regional Lymphadenitis: A Report of 60 Cases, Ann. Int. Med. **37**:697-713 (Oct.) 1952.

28. Clément, R.; Combes-Hamelle, A., and Meyer, B.: Forme oculaire de la lymphoréticulose bénigne dite "des griffes du chat," Bull. et mém. Soc. méd. **67**:1108 (Oct. 5) 1951.

Clinical Notes, New Instruments and Techniques

A DEHYDRATING JELLY TO CLEAR CORNEAL BEDEWING

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BEDEWING of the corneal epithelium may be observed in a number of pathologic processes. Generally, there are two prerequisites for its development in patients. There must be excessive hydration or fluid pressure in the underlying stroma; and the epithelial membrane must be intact and have relatively normal permeability characteristics. As a result of these two factors, fluid collects beneath and between the deep cells of the epithelium. This reduces the transparency of the cornea and gives its surface the classic "ground-glass" appearance of bedewing.

Bedewing of the corneal epithelium may occur in normal eyes when the corneal surface is maintained in contact with hypotonic solution, but this requires artificial circumstances, such as the wearing of a contact lens. Treatment of corneal bedewing in diseased eyes is directed primarily at the underlying pathology; however, there are many occasions, for example, in the management of patients with acute glaucoma, when the ophthalmologist wishes a transitory clearing of the bedewed epithelium so that proper biomicroscopic and ophthalmoscopic studies may be made. To accomplish this objective, he should consider both prerequisites to the development of edema of the corneal epithelium.

First, permeability of the epithelium can be increased by a combination of physical and chemical means which allow fluid to escape. This may be done without producing a physical break in the epithelium. Topical anesthesia increases the permeability of the epithelium. Furthermore, it condenses the precorneal film and thereby increases its osmotic pressure, a factor which favors removal of water from the epithelium. This condensation of the precorneal film results when the cornea and conjunctiva are anesthetized, because then lessened lacrimation and reduced blinking favor evaporation of fluid from the corneal surface.[1]

Further clearing of the bedewed epithelium can be effected by gentle massage of the anesthetized surface of the cornea. For this purpose, C. S. O'Brien has recommended the smooth, rounded tip of a glass rod.

Clearing of bedewed corneal epithelium can be hastened by instillation into the conjunctival sac of a highly hydroscopic collyrium. For this purpose, Cogan has

This study was made possible by funds given by Mrs. Helen Watzek and Sons.

From the Department of Ophthalmology, the University of Oregon Medical School.

1. Swan, K. C.; Pharmacology and Toxicology of the Cornea: Contemporary Concepts, Arch. Ophth. **41**:253-275 (March) 1949.

suggested glycerin.[2] Glycerin is readily available, is easily administered, and seems nontoxic when instilled into the conjunctival sac. Some decrease in epithelial bedewing results; however, the amount of clearing is reduced, and its duration is shortened by the lacrimation which glycerin is apt to induce through irritation of the conjunctiva. Some hyperemia and edema of the conjunctiva almost invariably result when glycerin is instilled. This may occur even when a local anesthetic has been used prior to the instillation of glycerin. It seemed probable, therefore, that the dehydrating effect of glycerin could be increased appreciably if, in its application, the collyrium could be confined to the cornea or a section of the cornea. This would be possible only if the viscosity of the glycerin could be increased. This objective has been reached quite satisfactorily by adding sodium carboxymethylcellulose. It is a white, fluffy solid, which is commonly used by pharmaceutical manufacturers to stabilize the emulsions and increase the viscosity of solutions.[3] Sodium carboxymethylcellulose dissolves in glycerin and greatly increases the viscosity of the latter without altering its transparency. Carboxymethylcellulose is hydroscopic and therefore may add to the dehydrating qualities of glycerin.

Carboxymethylcellulose should not be confused with methylcellulose. The latter, also, can be used to increase the viscosity of glycerin but is less satisfactory. Since the introduction of these methylcellulose compounds to ophthalmic therapeutics, both have been extensively used as ophthalmic vehicles, emollients, and substitutes for normal lacrimation in patients with keratoconjunctivitis sicca.[4] No ill effects upon the eye from their external application have been recorded in the literature. It appears, also, that hypersensitivity reactions to methylcellulose and carboxymethylcellulose must be very rare.

A satisfactory dehydrating jelly can be obtained by adding 2 gm. of sodium carboxymethylcellulose to 100 cc. of glycerin U. S. P.[5] This jelly then can be administered in a standard ophthalmic ointment tube with a fine tip. The jelly is very stable, but the tube should be kept capped when not being used. Samples prepared in 1950 still are being used in the University of Oregon Medical School hospitals and clinics. The samples also have remained sterile.

During the past two years, 2% sodium carboxymethylcellulose in glycerin has been administered without ill effects to the corneal surfaces of at least 200 patients. The following technique has been found to be most satisfactory: First, the corneal and conjunctival surfaces are anesthetized with two or three instillations of a topical anesthetic, such as 0.5% tetracaine hydrochloride. Then the lids are separated, and a small amount of the dehydrating jelly is placed on the corneal surface. The patient is advised not to wink. If the lids are held open by the physician, he should keep the lower lid in contact with the globe to prevent the jelly from sliding into the

2. Cogan, D. G.: Glycerin in Cornea: Clearing of Edematous Corneas by Glycerin, Am. J. Ophth. **26**:551 (May) 1943.

3. Brown, C. J., and Houghton, A. A.; The Chemical and Physical Properties of Carboxy-Methylcellulose and Its Salts, J. Soc. Chem. Ind., London **60**:254T-258T, 1941.

4. Swan, K. C.; Use of Methyl Cellulose in Ophthalmology, Arch. Ophth. **33**:378 (May) 1945.

5. To dissolve the carboxymethylcellulose completely, the glycerin should be heated in a water bath to at least 70 C.

lower fornix. The dehydrating jelly is thereby maintained on the corneal surface, and contact with the conjunctiva is avoided. Clearing of the epithelium generally is evident within a minute. This clearing can be increased by spreading the jelly on the cornea with a glass rod, at the same time gently massaging the epithelium. Appreciable clearing of stromal edema or large vesicles is not to be expected.

Examination of the eye can be begun within two or three minutes after application of the jelly. The substance is fairly transparent and tends to liquefy as it takes up water. It does not appreciably interfere with biomicroscopy but can be removed, if necessary, by irrigation. The degree and duration of the clearing of the corneal epithelium with this method vary with the individual patient, but they are more marked and prolonged than when glycerin is used alone, or even when it is combined with massage of the epithelium and local anesthesia. Moreover, the patient suffers less discomfort, because the jelly is largely confined to the cornea and relatively little reaches the conjunctiva.

AN AID IN OCULAR ANESTHESIA

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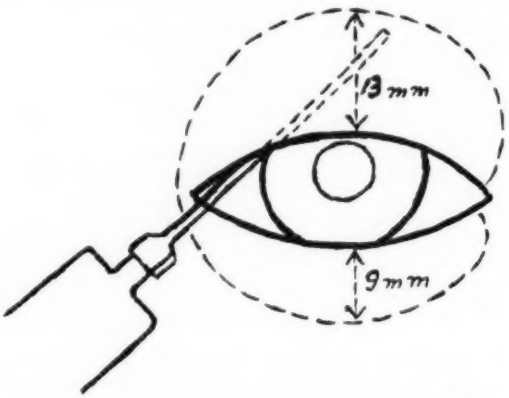
LOS ANGELES

ONE OF the most painful procedures during cataract surgery is the injection of a local anesthetic in the region of the superior rectus muscle preparatory to the placement of a control suture. Moreover, if this injection should fail to take effect, the passing of the needle through the muscle may easily become the most painful part of the entire operation.

The normal pressure of the upper lid against the eyeball prevents a sufficient concentration of topical anesthetic from remaining in the upper cul-de-sac even if a sufficient amount of the solution is placed there by drawing away the lid. According to Duke-Elder, the upper cul-de-sac is 13 mm. above the upper lid margin when the lids are open, and it is obvious that very little effect can be expected there from drops alone under ordinary circumstances.



Dimensions of the conjunctival sac, in millimeters, with the palpebral aperture open (after Duke-Elder). A needle, with a dull point, is shown in position when the cocaine-epinephrine mixture is deposited in the upper cul-de-sac.

The following procedure will as a rule provide a satisfactory answer to this problem.

A mixture consisting of 3 drops of 5% cocaine hydrochloride and 1 drop of 1:1,000 epinephrine hydrochloride is placed in a syringe, to which a long dull needle (e. g., lacrimal cannula) has been attached. After the usual anesthetic drops have been put in the eye and before the injection of procaine is begun, this mixture is "injected" high up in the cul-de-sac, as shown in the Figure. The solution is actually not injected, but is deposited there. Upon completion, the needle is withdrawn quickly to permit the lid to close against the globe. By the time akinesia and retrobulbar injections have been made, the top of the eyeball will as a rule be anesthetized sufficiently to permit the operation to proceed as usual without complaint from the patient.

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Registry of Interesting Cases

It is the purpose of this section to record cases on the basis of interest only. No complete review of the literature is attempted and no claim for priority is implied except when specifically stated. Publication of case reports is subject to the concurrence of the Editorial Board on the interesting aspects of the case. It is requested that the reports submitted be as brief as is consistent with inclusion of the pertinent facts, bear a title that is as specific as possible, be prefaced by a paragraph that states why the author feels the case is of interest and be accompanied with photographs whenever possible.

OPHTHALMOMYIASIS EXTERNA; LARVAL CONJUNCTIVITIS IN SOCKET

OCULAR myiasis, particularly the external form, is not uncommon in children of the tropical countries, but cases have been reported from Eastern and Western Europe. It is met with rarely in the United States. The symptoms of larval conjunctivitis are itching, burning, lacrimation and inflammation of the mucous membrane. Usually it is of no consequence, and the symptoms clear up promptly after removal of the maggots.

V. B., a 2-year girl from the rural section of this part of the country, was first seen on May 17, 1952. The child has been born at term. For a few months the parents had noticed the peculiar appearance of the child's right eye, which lately had become red and watery. She cried often. There were no eye diseases in the family, and the other five siblings have normal eyes.

Examination revealed photophobia in the right eye, with lacrimation, swollen lids, and ciliary injection of the eyeball. The cornea was of normal size; it was steamy; the anterior chamber was absent; the iris was brown-gray, hyperemic, and very atrophic; the pupil measured 5 mm. and was fixed and irregular. Tension was 3+. Behind the lens a whitish-gray mass was visible; no details could be made out because of cloudiness of the cornea. No consensual reaction was obtained to light. The left eye was normal.

A diagnosis of retinoblastoma with secondary glaucoma was made. The eye was enucleated on May 22, 1952. Pathologic examination revealed retinoblastoma.

Four days after enucleation the patient was discharged from the hospital; two days later the eye was dressed in the office; the parents failed to bring the patient back for the next arranged dressing. Seven days after the last dressing the patient was brought to the emergency room of the hospital at 1:30 a. m. because she had fever, was fretful, and the mother heard "maggots working in the eye."

The dressing was very soiled; it hung loose and had a foul odor. The temperature (rectal) was 102 F. After removal of the dressing three maggots wriggled out from the socket. The lids were swollen and inflamed. The conjunctiva of the socket was swollen, and an area of gray infiltration was visible at the apex. Cocaine solution was instilled to immobilize any other maggots which might be present in the socket, but no more were found. Because of the secondary infection with pyogenic micro-organisms, intramuscular administration of penicillin and streptomycin was instituted. Local treatment consisted of hot fomentations and irrigation of the socket with a solution of potassium permanganate. Within two days the temperature became normal, and the infiltration of the socket cleared in another few days.

The maggots were identified at the communicable disease center, Chamblee, Ga., as larvae of Phormia regina. This species is the black blowfly, belonging to the order of domestic flies. Its eggs hatch after a period of 8 to 30 hours. It is fundamentally a saprophytic fly, breeding in dead animal matter, and is common in cities and city dumps, as well as on the open range. In the South these flies become scarce during the hot summer months.

Concerning this species, James [1] writes:

Although this is one of the species that has been used in maggot treatment of wounds, it not uncommonly invades healthy tissue. It is a common sheep maggot in the Southwest and in certain localities may be more important as such than Callitroga americana during spring and fall months. Cases of traumatic dermal myiasis in man have been recorded; it has also been reported in enteric myiasis, although the record needs substantiation.

Maggots from socket.

It may be assumed that the child put her contaminated finger under the dressing and deposited the eggs of this fly on the lid or in the socket; however, as the dressing was loose, the fly had access to the lids, and it could have laid the eggs on them. The larvae, causing itching and burning of the conjunctiva, made the child rub her eyes, and a secondary infection of the socket with pyogenic micro-organisms followed, causing fever and infiltration of the apex of the socket.

1. James, M. T.: Flies that Cause Myiasis in Man, United States Department of Agriculture, Miscellaneous Publications, No. 631, 1948.

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Annual Reviews

OPTICS AND VISUAL PHYSIOLOGY

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BOOKS

ATTENTION should be called to a number of books published during the past year on optics, refraction, and visual physiology. Dr. Lancaster did not live to see the publication of his book [1] on optics and refraction, the writing of which had been his principal project for several years. This book is written in a very readable style, and the author has presented his approach to optical problems without the use of mathematics. As an introduction to the subject, it will be especially useful to the physician beginning his residency in ophthalmology. Although the title of Dr. Pascal's large book [2] on visual optics would make one believe it dealt only with special topics, it actually covers the field very completely. He has used new approaches to many old problems and has attempted to simplify the mathematics necessary to one's understanding of ophthalmic optics. In a small book of only 158 pages, Beach [3] has discussed a number of special topics in the principles of refraction that have been of particular interest to him. The book will also be of interest to ophthalmologists in general.

In physiological optics special attention is called to an excellent translation by Dr. Paul Boeder of the second edition of von Tschermak-Seysenegg's book, "Einführung in die physiologische Optik." The translation was sponsored by the American Committee on Optics and Visual Physiology of the American Medical Association.[4] The portion of the book pertaining to the space sense is in one sense classic. The portion dealing with the light sense, and especially color theory, while of considerable historical interest, is much outdated. Dr. Linksz, a one-time pupil of von Tschermak-Seysenegg, has published the second volume of his series. This large volume on visual physiology [5] encompasses the light and space senses and the basis of ocular motility involved in fusion. The book is conversational in style, up-to-date, and fairly comprehensive. Controversial theories pertaining to various phenomena in visual perception are discussed in the text. Moncrieff [6] has proposed

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1. Lancaster, W. B.: Refraction Correlated with Optics and Physiological Optics, and Motility Limited to Heterophoria, Springfield, Ill., Charles C Thomas, Publisher, 1952

2. Pascal, J. I.: Studies in Visual Optics, St. Louis, C. V. Mosby Company, 1952.

3. Beach, S. J.: Principles of Refraction, St. Louis, C. V. Mosby Company, 1952.

4. von Tschermak-Seysenegg, A.: Introduction to Physiological Optics, translated by P. Boeder, Springfield, Ill., Charles C Thomas, Publisher, 1952.

5. Linksz, A.: Physiology of the Eye, New York, Grune & Stratton, Inc., 1952, Vol. 2.

6. Moncrieff, M. M.: The Clairvoyant Theory of Perception: A New Theory of Vision, New York, Harper & Brother, 1952.

a rather vague theory that visual perception is a specialized form of clairvoyance, that is, of extrasensory perception. From this reviewer's point of view, the book need not be taken seriously. For those who would be interested in obtaining a well-rounded survey of visual physiology (and the psychophysiology of vision), the three chapters (22, 23, and 24) in the handbook of experimental psychology edited by Stevens [7] and written by Judd, Graham, and Bartley, respectively, are recommended. Ittelson [8] has prepared a monograph to describe the many demonstrations devised by Ames to illustrate the influence of certain factors or clues involved in visual spatial localization.

Judd [9] has written an excellent book on color, a book which could be useful to the businessman and to the scientist alike. Dr. Judd is certainly one of the authorities most competent to write such a book, and in general he has succeeded in presenting a readable and well-rounded story of color, color measurement, and color anomalies.

Murray [10] has published a revised and enlarged edition of his book on color, originally published in 1939. The book presents basic facts of all aspects of color perception and is suitable for an introduction to the subject.

VISUAL SCREENING TESTS

A study [11] was made of the visual defects of the first- and sixth-grade school children in St. Louis, in which the screening procedures used were: the ordinary Snellen test card, the Massachusetts Vision test, the Telebinocular, the Ortho-Rater, the Sight-Screener, and a near-vision test. These tests were conducted by nurses, technicians, and teachers. The results on 1,215 students showed that on the basis of machine tests the number of children incorrectly referred to the ophthalmologist for further examination was as large as that correctly referred. None of the tests provided more than a rough screening procedure. With the Snellen chart, even when used by a teacher, there were fewer incorrectly referred children, and the test could be completed in a shorter time. The particular test used for screening must depend on the purpose of the test. In another, but similar, study the Massachusetts Vision test was evaluated in a school population of 1,575 students by Yasuna and Green.[12] As a result of their study, they recommend that the 20/20 line on the Snellen chart not be used in the early grades and that the 20/30 line be used as the basis for failure to pass the test. To screen for hyperopia, a + 2.25 D. sphere before each eye is recommended. The authors also recommend that heterophoria tests be eliminated, since the data obtained are unreliable. On the other hand, Morgan and

7. Stevens, S. S., Editor: Handbook of Experimental Psychology, New York, John Wiley & Sons, Inc., 1951.

8. Ittelson, W. H.: The Ames Demonstrations in Perception, Princeton, N. J., Princeton University Press, 1952.

9. Judd, D. B.: Color in Business, Science, and Industry, New York, John Wiley & Sons, Inc., 1952.

10. Murray, H. D., and Spencer, D. A.: Colour in Theory and Practice, London, Chapman & Hall, Ltd., 1952.

11. Crane, M. M.; Scobee, R. G.; Foote, F. M., and Green, E. L.: Study of Procedures Used for Screening Elementary School Children for Visual Defects: Referrals by Screening Procedures vs. Ophthalmological Findings, Sight-Saving Rev. **22**:141-153 (Fall) 1952.

12. Yasuna, E. R., and Green, L. S.: An Evaluation of the Massachusetts Vision Test for Visual Screening of School Children, Am. J. Ophth. **35**:235-240 (Feb.) 1952.

collaborators,[13] who tested 1,200 children in the Toronto public schools, consider that 20/30 visual acuity, as measured by the illiterate E, can be regarded as normal only for the kindergarten age group. For grades one to eight vision of 20/20 should be regarded as normal. In an intensive study, Granström [14] measured the visual functions in 1,254 applicants to the Swedish Air Force, all between the ages of 18 and 25. Of these, 29% had visual acuity of 20/20 or better, and 0.5% had a visual acuity of 20/10. Orthophoria was found in 49%, esophoria in 23%, and exophoria in 22%. Poor stereoscopic vision was found in 0.2% of the subjects, and of these 46% showed orthophoria. Motility, light sense and refractive errors were also tabulated.

REFRACTION

A study of the refractive error of the eyes of 1,000 newborn infants measured by retinoscopy with atropine cycloplegia is reported by Cook and Glasscock.[15] Nearly 75% of the infants showed hyperopia up to 12 D., but the median value for the hyperopia was between 2 and 3 D., and for nearly 90% of the babies it was less than 6 D. About 25% of the infants were myopic up to 12 D., but the distribution according to degree of myopia was very similar to that for the hyperopia. Astigmatism occurred in nearly 40% of the infants. Hirsch [16] studied statistically the changes in refraction of more than 9,500 school children between the ages of 5 and 14 years. These children were taken at random. The refractive errors were measured by the retinoscope with accommodation relaxed. The average refractive error was a hyperopia, but this average decreased steadily (linearly) but slowly with age. There was, of course, little change in refractive error in the majority of children. The decrease in the mean refractive error was from +0.90 to +0.40 D. for girls and from +0.75 to +0.30 D. for boys. The decrease in the median refractive error was a little less. These data give some basis for prediction of change in hyperopia in this age span. The distribution curve is skewed toward hyperopia at about 9 or 10 years of age and then is skewed toward myopia with increased age. Yamamoto [17] discusses the data for the relation between refractive error and growth which he obtained from 452 students. These data show a significant correlation of body sitting weight and chest girth with refractive error. However, within the same age group this correlation was not significant. Fraňa and Vlček [18] report that the incidence of myopia is high among leather cutters and that this myopia increases with age and duration of employment. No connection with health or outside activities could be found. To ascertain the extent to which astigmatism of the cornea might change

13. Morgan, A. L.; Crawford, J. S.; Pashby, T. J., and Gaby, J. R.: A Survey of Methods Used to Reveal Eye Defects in School Children, Canad. M. A. J. **67**:29-34 (July) 1952.

14. Granström, K. O.: Eyes With Normal Visual Acuity Without Glasses: An Analytical Study, Acta ophth. **30**:107-114, 1952.

15. Cook, R. C., and Glasscock, R. E.: Refractive and Ocular Findings in the Newborn, Am. J. Ophth. **34**:1407-1413 (Oct.) 1951.

16. Hirsch, M. J.: Changes in Refraction Between the Ages of 5 and 14: Theoretical and Practical Considerations, Am. J. Optom. **29**:445-459 (Sept.) 1952.

17. Yamamoto, Y.: Myopia and Physical Constitution, Supplementary Report No. 1, Acta Soc. ophth. (Japan) **56**:30-33 (April) 1952; Supplementary Report No. 2, ibid. **56**:33-35 (May) 1952.

18. Fraňa, G., and Vlček, T.: Změny refrakce u sekáčů kůže, Pracovní lékarstvi (Praha) **3**:72-85, 1951.

with age, from that "with the rule" to that "against the rule," Phillips [19] analyzed his clinical records and found that this change was greatest in the 30 to 40 age group and that after the age of 60 no change occurred.

For determining astigmatism, an "arrow" type rotatable dial test is described by Raubitschek.[20] Two curved lines (parabolas) form a narrow V-like, or arrowhead, figure. This dial is slowly rotated until the subject reports that both limbs of the arrow are equally black. This position, which gives the axis of astigmatism, proves to be a sensitive test. An approximate cylinder lens is then placed before the eye but not on the axis indicated by the dial. The lines then are no longer equal in darkness, but the dial position will be changed until they are. The difference in positions of the dial and the power of the cylinder provide the data from which true astigmatic correction can be obtained from a table. In a comparative study of the astigmatism determined subjectively and that measured by the ophthalmometer, Paycha [21] found that lenticular (not corneal) astigmatism for emmetropes and myopes tended to be —0.50 D. at axis 90 degrees. However, for hyperopic subjects it tended to be —0.50 D. at axis 180 degrees. In astigmatism at oblique axes there appears to be no relationship between the measurements on the ophthalmometer and the total astigmatism. Borsello [22] found that the curvature of the cornea was affected by the advancement of the medial and lateral rectus muscles. The astigmatism of all patients was permanently changed in amounts varying from 0.2 to 1.1 D. Along a similar vein, Pavišić [23] concluded that pterygium changes the curvature of the cornea, there being on the average a flattening of 1.5 D. in the horizontal meridian in 70% of cases. In the vertical meridian, also, there was a slight flattening in 32% of cases, or a slight increase in curvature. Rumpelhardt [24] in a large number of cases (more than 50,000), found a higher incidence of myopia in eyes with maculae corneae than in those without, the incidence being higher the greater the myopia. The ratio of numbers of cases in the two categories for a myopia of —1 D. was 1.43 (43%), but for a myopia of —10 D. the ratio was 3.5. The ratio for hyperopic eyes was between 1.2 and 1.6, with no trend toward higher degrees of hyperopia.

LENSES

In a short article, Miles [25] discusses corrected ophthalmic spectacle lenses, pointing out their particular value for high refractive errors. A corrected type of spectacle lens should be available for patients with marked astigmatism, but in this condition

19. Phillips, R. A.: Changes in Corneal Astigmatism, Am. J. Optom. **29**:379-380 (July) 1952.

20. Raubitschek, E.: The Raubitschek Arrow Test for Astigmatism, Am. J. Ophth. **35**: 1334-1339 (Sept.) 1952.

21. Paycha, F.: Des différences entre les résultats de l'ophtalmometrie et de la méthode de Donders, Arch. opht. **12**:296-299, 1952.

22. Borsello, G.: Variazioni delle curvature corneali consecutive ad avanzamento del muscolo retto esterno od interno, Rass. ital. ottal. **21**:55-62 (Jan.-Feb.) 1952.

23. Pavišić, Z.: Untersuchungen über Refraktion und Astigmatismus der Hornhaut beim Pterygium, Ophthalmologica **124**:157-165 (Sept.) 1952.

24. Rumpelhardt, K.: Untersuchungen über die Beziehungen zwischen Myopie und Hornhautnarben, Klin. Monatsbl. Augenh. **120**:397-403, 1952.

25. Miles, P. W.: Importance of Corrected Curves in Spectacle Lenses, Am. J. Ophth. **35**:1320-1328 (Sept.) 1952.

bitoric lenses would be required. Jensen [26] showed that it was impossible by change of base curve to make spherical spectacle lenses free from astigmatism over the complete field of view, for all object distances and for all the zones of a given lens. Working on the same problem, Roos [27] has made considerable calculation for the astigmatism for rays passing through the peripheral parts of spherical spectacle lenses. For small lenses there is little astigmatism for object points as near as 1 meter. The lenses with larger diameters (50 mm.) cannot be free from astigmatism in the most peripheral parts with only two surfaces to work from. However, Roos expresses the belief that a tolerance should be set for astigmatic errors of almost 0.25 D. It is easier to design lenses which do not admit larger astigmatic errors than this. Kühl,[28] however, believes that the limiting astigmatic error for an oblique ray should be 0.06 D. for true "point" imagery (*punktuellen Abbildung*).

On the basis of a previously published analytical theory, [29] Ogle [30] describes in practical terms the distortion of the image caused by ophthalmic prisms. It is shown that increasing the base curve of the prism reduces the curvature of the images of vertical lines (but never eliminates it), reduces the increase of magnification toward the apex of the prism (and eliminates it for a particular base curve, for a given distance of prism from the eye), but increases the slanting of the images of lines parallel to, but above and below, the base-apex meridian. The distortion of ophthalmic prisms is important in the problem of disturbed stereoscopic spatial localization due to such prisms.

Rosett [31] derives the familiar formula for the angular magnification of ophthalmic lenses and applies it to cases of anisometropia and suspected aniseikonia. Blum [32] discusses his experiences with nine subjects with unilateral aphakia whose vision had been corrected with contact lenses. Six reported simultaneous macular perception and stereoscopic vision. However, most of the patients had diplopia or strabismus and were unable or unwilling to wear the contact lenses. To this reviewer the results are not convincing that normal binocular vision was obtained in any but several patients. Admittedly, residual aniseikonia was the major difficulty experienced. The use of pinhole contact lenses is described by Freeman [33] for use in patients with albinism, dyscoria and polycoria, and subnormal vision. The lens has an artificial iris for cosmetic purposes. Such a pinhole contact lens with a 2 mm. aperture can be used in subjects with presbyopia to give a universal focus. The

26. Jensen, H.: Astigmatism of Spherical Spectacle Lenses and Point-Focal Imagery, Optik (Weimar) **9**:193-205, 1952.

27. Roos, W.: Über die Definition der punktuellen Abbildung bei sphärischen Brillengläsern, von Graefes Arch. Ophth. **151**:513-531 (June) 1951.

28. Kühl, A.: Über die Definition der punktuellen Abbildung, von Graefes Arch. Ophth. **152**:545-550 (March) 1952.

29. Ogle, K. N.: Distortion of the Image by Prisms, J. Optic. Soc. America **41**:1023-1028 (Dec.) 1951.

30. Ogle, K. N.: Distortion of the Image by Ophthalmic Prisms, A. M. A. Arch. Ophth. **47**:121-131 (Feb.) 1952.

31. Rosett, B.: Magnifying Effects of Spectacle Lenses, Am. J. Optom. **29**:341-346 (July) 1952.

32. Blum, J. D.: Aphakie unilatérale et verres de contact, Ophthalmologica **123**:284-290 (April-May) 1952.

33. Freeman, E.: Pinhole Contact Lenses, Am. J. Optom. **29**:347-352 (July) 1952.

making of these lenses requires great skill, for they must fit accurately and the pinhole must be well centered. The haze and visual halos experienced by those who wear contact lenses due to the scattering and diffracting characteristics of the optical parts involved have been studied by Finkelstein.[34]

NIGHT MYOPIA

The literature pertaining to the cause of night myopia (a condition in which the eye apparently becomes increasingly myopic under conditions of lower and lower illumination levels) has been well, but briefly, reviewed by Knoll[35] from a comprehensive bibliography. While the experiments of Koomen, Scolnik, and Tousey[36] showed convincingly that night myopia is due to the increasing effect of spherical aberration, Otero and Aguilar[37] express the belief that the phenomenon is due to an actual accommodation of the eye, which they believed they had measured by photographing the third catoptric image of two light sources from the eye in darkness. In a separate experiment, they could not find any major influence of spherical aberration in five subjects. Just recently Koomen, Scolnik, and Tousey[38] have repeated Otero's experiment of photographing the third catoptric (Purkinje) image and could find no evidence of an accommodation in dim illumination or in complete darkness. Moreover, Bouman and van den Brink[39] have recently determined the visual acuity through a telescope for various focal settings for each eye separately, using various test objects, illuminations, pupil sizes and directions of fixation. The results showed that to a large extent night myopia was due to spherical aberration, a result that tended to confirm the conclusions of Koomen and associates.

ACCOMMODATION

When a young emmetrope looks from a distant object to another object at a distance of 1 meter, the eye is said to accommodate 1 D. Pascal[40] pointed out that when a corrected ametrope accommodates for the same two points, the actual accommodative change of the crystalline lens is more or less than 1 D. depending on whether the correcting lens is plus or minus, on the strength of that lens, and on its distance from the eye. Simple approximate formulas are discussed for calculating the change in "accommodative units" of the emmetropic eye. An important application of this consideration is the accommodation of the two eyes in corrected

34. Finkelstein, I. S.: Biophysics of Corneal Scatter and Diffraction of Light Induced by Contact Lenses, Am. J. Optom. **29**:185-208 (April) 1952; **29**:231-259 (May) 1952.

35. Knoll, H. A.: A Brief History of "Nocturnal Myopia" and Related Phenomena, Am. J. Optom. **29**:69-81 (Feb.) 1952.

36. Koomen, M.; Scolnik, R., and Tousey, R.: A Study of Night Myopia, J. Optic. Soc. America **41**:80-90 (Feb.) 1951.

37. Otero, J. M., and Aguilar, M.: Accommodation and Night Myopia, J. Optic. Soc. America **41**:1061-1062 (Dec.) 1951.

38. Koomen, M.; Scolnik, R., and Tousey, R.: Measurement of Accommodation in Dim Light and in Darkness by Means of the Purkinje Images, J. Optic. Soc. America **43**:27-31 (Jan.) 1953.

39. Bouman, M. A., and van den Brink, G.: On Night Myopia, Ophthalmologica **123**:100-113 (Feb.) 1952.

40. Pascal, J. I.: Scope and Significance of the Accommodative Unit, Am. J. Optom. **29**: 113-128 (March) 1952.

anisometropia, where the "accommodative units" would be unequal. Graff [41] also discusses this problem when a near-vision correction, as well as the distance correction, is involved in fixating from a distant to a near object. Pascal [42] also discusses the reverse problem of determining the power of the corrective lens that will replace a given amount of accommodation.

The observation first reported by Gullstrand that an eye turns slightly outward on change of accommodation from a distant to a near object has been confirmed and measured by de Leonibus.[43] While a distant object point is fixated, a near object point 25 cm. from the eye is aligned with the distant object. A small mark or blood vessel near the limbus of the cornea is observed by the experimenter through a micrometer-telescope. When the observer changed accommodation from the distant to the near object without change in fixation axis, the experimenter observed a slight outward (temporal) rotation of the eye in 14 of 20 subjects. The magnitude of the rotation varied from 10 to 74 minutes of arc.

Fletcher [44] tried to determine with the Fincham coincidence optometer whether there could be an astigmatic accommodation, but his results were entirely negative. LeGrand [45] found that certain emmetropic subjects who had been made artificially myopic (by placing a plus lens before one eye), when making an effort to see clearly, exhibited a "negative" accommodation as measured by the retinoscope on the other eye, even as much as 2 or 3 D. With cycloplegia, the basic refractive error was little changed, but the negative accommodation was reduced.

VISUAL ACUITY

The problem of the distribution of the light energy within the retinal image and the subsequent determination of limiting resolving power or limiting visual acuity is still being attacked. Arnulf and associates [46] determined by a micrometer the apparent size of the disk of light confusion from a point source of light on the retina for various levels of illumination. From the data obtained they calculated the distribution of light within the image. They conclude that diffusion, or scattering, of light within the retinal structure is the important phenomenon determining that distribution. Junés [47] concludes that the pigmented epithelium between the cones regulates the distribution of light effective in the cones. This will depend on the height of the pigment epithelium surrounding the cones and, therefore, will vary with the intensity of the light falling on the retina and its adaptation. O'Brien [48]

41. Graff, T.: Die Akkommodation beim Sehen durch das Nahbrillenglas, Klin. Monatsbl. Augenh. **121**:205-215, 1952.

42. Pascal, J. I.: Lens Replacement of Accommodative Activity, Ophthalmologica **123**: 394-396 (June) 1952.

43. de Leonibus, F.: Akkommodative Richtungsänderung der Augenachse, Ophthalmologica **123**:15-19 (Jan.) 1952.

44. Fletcher, R. J.: Astigmatic Accommodation, Brit. J. Physiol. Optics **8**:193-224, 1951.

45. LeGrand, Y.: The Presence of Negative Accommodation in Certain Subjects, Am. J. Optom. **29**:134-136 (March) 1952.

46. Arnulf, A.; Dupuy, O., and Flamant, F.: Répartition de la lumière dans l'image d'un point lumineux sur la rétine, Compt. rend. Acad. sc. **233**:974-976 (Oct. 22) 1951.

47. Junés: Action de l'éclairage sur l'acuité visuelle des cones fonctionnant en vision directe: Role de l'épithélium pigmentaire, Ann. ocul. **185**:209-237 (March) 1952.

48. O'Brien, B.: Vision and Resolution in the Central Retina, J. Optic. Soc. America **41**:882-894 (Dec.) 1951.

states that the shape of the cones prevents the spread of rays beyond the plane of focus of the image. On the basis that the retinal receptors do absorb light directionally, di Francia and Ronchi [49] conclude that the receptors should also scatter the incident light directionally. A slit was made in the sclera of a freshly excised eye extending from near the limbus of the cornea to the equator of the globe. Parallel incident light was sent into the eye on the optic axis, and the intensity of the scattered light was measured at different azimuth angles through the slit. It was found that the intensity of the scattered light was five times as great at 70 degrees from the pole of the cornea as at 26 degrees, thus showing definitely a directional effect.

By using two pinholes, the light through which came from a single pinhole or slit (thus making the light coherent), Arnulf and associates [50] studied the diffraction fringes. The separation of the two pinholes could be altered. With a small pupil (1.6 mm.), the optical irregularities of the eye media were eliminated, and the diffraction fringes caused by the pinholes could be studied. The separation of the two pinholes was adjusted for minimal resolution when the pupil was centered (1) on a bright and (2) on a dark diffraction band. The behavior of the two Airy disks corresponding to the images of the pinholes was very different in the two cases. A somewhat similar method was used by O'Brien and Miller [51] to determine the resolution of the retina itself, and hence the separation of the cone receptors in the fovea, except that slits were also used.

As is well known, the resolving power of the eye for two parallel lines depends on the length of those lines up to a certain limit in length. Otero and associates [52] advance a hypothesis based on the quantum theory of vision to explain this.

The change in visual acuity using a grating test object, with size of artificial pupil for different luminance levels, was investigated by Leibowitz.[53] In his study, however, he adjusted the luminance of the test object so as to keep the apparent luminance of the reference field constant. As pupil size increases, the acuity increases rapidly at first, then more slowly until a maximum is reached. If the pupil size can then still be increased, the acuity actually decreases. Aberrations of the eye become a factor of increasing importance as the pupil increases in size. Von Bahr [54] related the depth of focus of the eye to acuity by measuring the resolving ability of the eye for two short vertical lines as the image on the retina is blurred by power lenses. He found that a range for blue light of 1.5 to 2.0 D. was possible in some subjects. However, conclusions as to the effect of illumination and chromatic aberration were uncertain because of the anomalous result that monochromatic light gave better depth of focus than did daylight of the same intensity.

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49. di Francia, G. T., and Ronchi, L.: Directional Scattering of Light by the Human Retina, J. Optic. Soc. America **42**:782-783 (Oct.) 1952.

50. Arnulf, A.; Dupuy, O., and Flamant, F.: Phénoméne de diffraction dans l'image de deux points lumineux en lumiére cohérente, Compt. rend. Acad. sc. **234**:1273-1275 (March 17) 1952.

51. O'Brien, B., and Miller, N. D.: Resolving Power of the Retina for Chromaticity Contrast and for Luminance Contrast, abstracted, J. Optic. Soc. America **42**:289 (April) 1952.

52. Otero, J. M.; Aguilar, M., and Solis, M.: Visual Acuity: Its Dependence on the Length of the Test Objects, An. real. Soc. españ. fis. y quim. **47**:289-294 (Nov.-Dec.) 1951.

53. Leibowitz, H.: Effect of Pupil Size on Visual Acuity for Photometrically Equated Test Fields at Various Levels of Luminance, J. Optic. Soc. America **42**:416-422 (June) 1952.

54. von Bahr, G.: Studies on the Depth of Focus of the Eye, Acta ophth. **30**:39-44, 1952.

In a comparative study of the effectiveness of the Landolt ring and a specially designed chart of 10 letters, Sloan and associates [55] found the two tests essentially equivalent. However, in a comparison with the Bausch and Lomb checkerboard target, significant differences were found, in that with small uncorrected astigmatic errors special additonal cues aided in the recognition of out-of-focus checkerboard squares. The value of the Landolt ring as a measurement of absolute acuity was also investigated by Schober.[56] Errors when gaps were seen in the opposite end of the diameter of the ring were found to occur too frequently, although the number of errors was least when the gap was in the vertical and horizontal meridians. A psychological explanation is suggested for these errors.

In a series of tests for visual acuity using a letter chart at a true distance and the equivalent optically simulated distance by lenses on the Ortho-Rater, Altman and Rowland [57] found the correlation to be so high that they concluded that visual acuity tests in which the visual distance is simulated give the same results as tests made at the true distance, provided illumination, contrast, and other factors are made the same. This result has a bearing on reports that, in a simulated distance test, by the target's appearing nearer there is provided a stimulus for accommodation.

De Leonibus [58] found that when a stenopaic opening was moved back and forth in front of the pupil of the eye, the visual acuity remained fairly constant so long as the opening was maintained near the central part of the cornea, but decreased rapidly when displaced toward the edges of the pupil. This effect might be attributed to spherical aberration (Kincaid and Blackwell [59]) or other off-axis irregularities in the ocular media. When words were presented on a screen tachistoscopically in different parts of the field of vision, Mishkin and Gorgays [60] report that they found recognition of words in the right side of the field better than that of words in the left side, and of those below the fixation point better than that of those above. This difference is attributed to the selective retinal training that occurs because of our accustomed pattern of reading.

The experimental work of Weston [61] showed that the ability to perform a given visual task decreased with the age of the subject. When the illumination of the task was increased, there was an increase with increasing age. However, it was not possible to compensate for the loss of performance with age with an increase in

55. Sloan, L. L.; Rowland, W. M., and Altman, A.: Comparison of 3 Types of Test Target for the Measurement of Visual Acuity, Quart. Rev. Ophth. **8**:4-16 (March) 1952.

56. Schober, H.: Investigation of the Applicability of Landolt's C as a Standard in Visual Acuity Measurements, Optik (Weimar) **9**:225-235, 1952.

57. Altman, A., and Rowland, W. M.: Measures of Acuity with Optical Simulation of Distance, Quart. Rev. Ophth. **8**:1-3 (March) 1952.

58. de Leonibus, F.: Influenza del decentramento pupillare sull'acutezza visiva, Ann. ottal. m. clin. ocul. **77**:62-70, 1951.

59. Kincaid, W. M., and Blackwell, H. R.: Irregularities of Retinal Illumination Resulting from an Artificial Pupil, abstracted, J. Optic. Soc. America **42**:873 (Nov.) 1952.

60. Mishkin, M., and Gorgays, D. G.: Word Recognition as a Function of Retinal Locus, J. Exper. Psychol. **43**:43-48 (Jan.) 1952.

61. Weston, H. C.: Illumination and the Variation of Visual Performance with Age, Commission international de l'eclairage, 12th Meeting, 1951, Vol. 2, paper C, 1952, New York Central Bureau C. I. E.

illumination. Brožek and Simonson [62] sought by extensive tests and statistical analysis to find reliable indices of visual performance and fatigue under different illuminations. None of these were entirely satisfactory, but as an over-all result it would appear that no importance can be ascribed to differences in the effect of spectral composition of the illumination on the visual efficiency or on fatigue. Somewhat similarly, Solandt and associates [63] measured the visual acuity under seven different white-light illuminations, all at 10 foot-lamberts, but of very different spectral distributions. No significant effect was found. Tinker [64] calls attention to the fact that in interpreting data on illumination and visual acuity the method of plotting data against the logarithm of illumination should not be used, since it is very misleading. The small gains in visual acuity at higher levels of illumination are not significant. A simple curve obtained by plotting the visual acuity against the level of illumination would be better interpreted.

From time to time, patients with uncorrected myopia are reported who for short periods have "flashes" of clear vision. Giglio [65] reports on four such persons and proposes the hypothesis that the ability to have flashes of clear vision results not from practice or interpretation of blur circles, but from changes on the anterior corneal surface, "which provide multiple auxiliary optical systems of small aperture," such as some type of liquid film on the corneal surface. Marg [66] reviewed much of the literature, especially that of Bates and followers, on this problem and was able to obtain five subjects for study. During such flashes of clear vision (during which the visual acuity may not actually be better than 20/50), he was not able to find any evidence of change of refractive state of the eye (no negative accommodation). Of 100 clinic subjects with ages between 14 and 40 years, only 1 demonstrated good transient vision. Marg suggests that the improvement of the perceptual images was due to training interpretation of image blur. Fry [67] also discusses this phase of training of myopes to "see more clearly."

The most controversial topic at present is whether yellow-tinted spectacle lenses, by reducing glare, are an aid in driving at night. Several papers, for example, that of Broom and Cole,[68] present data showing that these lenses do reduce glare and that this reduction is "achieved without significant loss in visibility." However, these authors measured only reduction of glare with General Electric foot-candle meters and made no measurements on thresholds of visibility or on the possible reduction

62. Brožek, J., and Simonson, E.: Visual Performance and Fatigue Under Conditions of Varied Illumination, Am. J. Ophth. **35**:33-46 (Jan.) 1952.

63. Solant, D. Y.; White, R. E., and Rosen, P. S.: Visual Importance of the Spectral Components of White Light, Illum. Engineer., N. Y. **47**:435-444 (Aug.) 1952.

64. Tinker, M. A.: Interpretation of Illumination Data, Am. J. Optom. **29**:293-300 (June) 1952.

65. Giglio, E. J.: Visual Acuity Under Special Circumstances, Am. J. Optom. **29**:647 655 (Dec.) 1952.

66. Marg, E.: "Flashes" of Clear Vision and Negative Accommodation with Reference to the Bates Method of Visual Training, Am. J. Optom. **29**:167-184 (April) 1952.

67. Fry, G. A.: Psychological Factors in Visual Acuity, Am. J. Optom. **29**:624-630 (Dec.) 1952.

68. Broom, M. E., and Cole, O. N.: Automobile Headlighting in Night Driving, Optometric Weekly **43**:409-411 (March 13) 1952.

of visual acuity through the tinted lenses. A number of writers [69] are taking issue with the statements made in advertising promotion for these lenses and insist, contrariwise, that wearing such lenses actually increases the hazard of driving at night. The apparent increase in clarity reported by some wearers is laid to an optical illusion. The lenses do not decrease glare "because the brightness relationship between one's own headlights and oncoming headlights is not changed." Such lenses also impair the ability of some 2% of the population to distinguish traffic lights. A statistical study of the effect of such lenses will probably be published within the coming year. Bierman [70] concludes from a study that began with 136 marksmen, all of whom had 20/20 visual acuity that yellow lenses do not improve marksmanship in shooting. The impression of a clearer image was deemed to be psychological. Some of the men liked the lenses; others did not.

Ohm [71] again reports on the objective measurement of visual acuity by means of optokinetic eye movements. In this study he used a photographic method of recording the nystagmoid eye movements and used his method of inhibiting the nystagmus with his special fixation mark. The smallest mark which will inhibit the movements is taken as a measure of the visual acuity. However, Schumann [72] has reviewed these various methods and describes in particular the method of Guenther, in which the optokinetic eye movements were stimulated by a black and white checkerboard pattern of several sizes on a rotating cylinder. Here optokinetic eye movements will occur only so long as the subject can resolve the pattern on the rotating drum.

LIGHT SENSE—THRESHOLDS

In a discussion on the need of protective dark glasses, Hoffmann [73] maintains that while ultraviolet radiation is for the most part prevented from reaching the retina in young people, in older ones more ultraviolet radiation is transmitted, and in those more than 60 years of age it may cause injury to the retina. This view regarding the effects of ultraviolet radiation is not generally held. Even that the exposure to ultraviolet radiation of 290 to 365 mμ can increase the light thresholds of normal eyes, as reported by Zigler, Wolf, and King,[74] is vigorously denied by Wald.[75] He insists, from a review of available data, that light rays of wave length shorter than 300 mμ are absorbed by the cornea and do no damage and that those longer than 300 mμ, and up to 400 mμ, are absorbed by the lens. He concludes "that ultraviolet

69. Richards, O. W.: Vision at Levels of Night Road Illuminations, in Night Visibilty, Bulletin 56, of the Highway Research Board, National Academy of Sciences, National Research Council Publication No. 236, 1952, pp. 36-65. Another Warning Against Tinted Lenses at Night, Editorial, Sight-Saving Rev. **22**:223 (Winter) 1952.

70. Bierman, E. O.: Tinted Lenses in Shooting, Am. J. Ophth. **35**:859-860 (June) 1952.

71. Ohm, J.: Über die Anwendung bewegter Spiegelmarken in Verbindung mit dem optokinetischen Nystagmus zwecks objektiver Sehschärfenbestimmung, Klin. Monatsbl. Augenh. **120**:144-150, 1952.

72. Schumann, W. P.: Objective Determination of Visual Acuity on the Basis of the Optokinetic Nystagmus, Am. J. Optom. **29**:575-583 (Nov.) 1952.

73. Hoffmann, W.: Schutzbrillenindikation und neue Glasarten, Klin. Monatsbl. Augenh. **120**:113-126, 1952.

74. Zigler, M. J.; Wolf, E., and King, E. S.: Influence of Surround Brightness and Short Wave Components of Radiation on Dark Adaptation, J. Optic. Soc. America **41**:354-359 (May) 1951.

75. Wald, G.: Alleged Effects of the Near Ultraviolet on Human Vision, J. Optic. Soc. America **42**:171-177 (March) 1952.

radiations which might do harm to the retina do not reach it; those which do reach the retina do it no harm, and can at most be seen." On the other hand, Peckham and Harley [76] state that excessive exposure to bright light without sunglasses can reduce the retinal sensitivity of the eye as much as 50%, and that subsequent dark adaptation may be retarded for days following that exposure. Such a loss of retinal light sensitivity could lead to industrial and automobile accidents under conditions of low illumination. The necessity of wearing dark glasses under these surroundings of high brightness is great. Sunglasses that transmit 10 to 12% of the incident light are recommended. Peckham [77] also suggests that the changes in dark adaptation attributed by Zigler, Wolf, and King [74] to ultraviolet radiation may, instead, be due to visible light within highly effective regions of the spectrum.

Hsia and Graham [78] have redetermined cone spectral sensitivity curves for five persons, using the accurate instrumentation developed and used by Hecht, Shlaer, and Pirenne for their quanta determinations. Among other reasons for the redetermination was the suspicion that the presently accepted luminosity curve was in error. The new data obtained by threshold measurements show a maximal sensitivity near 550 mμ, but the curve shows two points of abrupt change in direction, near 460 and 600 mμ, respectively. These changes have been suggested in the results of other investigators.

Davy [79] presents data which show that the rod (15 degrees in the peripheral retina) threshold to light perception for a stimulus field of 2 minutes of arc was unaffected by breaking up the light stimulus into separate flashes if those flashes occurred within a critical duration of 0.1 second. Brindley [80] has recently shown that the visual effect of a light stimulus is dependent on the product of the intensity and the time of exposure (Bunsen-Roscoe law) even when the times of exposure are in the range of 1.54×10^{-3} to 4.11×10^{-7} second.

DARK ADAPTATION

The dark adaptation threshold was measured by Ferguson and Sharpley [81] on a large number of miners and nonminers. They found that the dark adaptation threshold for miners was generally higher than that for nonminers and that it is even higher still for miners with nystagmus.

The normal distribution curve of threshold intensities for intermittent light for the central to the peripheral part of the retina was investigated by Lange.[82] He con-

76. Peckham, R. H., and Harley, R. D.: Effect of Sunglasses in Protecting Retinal Sensitivity, Am. J. Ophth. **34**:1499-1507 (Nov.) 1951.

77. Peckham, R. H.: A Proposed Method for Predicting Light Adaptation, J. Optic. Soc. America **42**:65 (Jan.) 1952.

78. Hsia, Y., and Graham, C. H.: Spectral Sensitivity of the Cones in the Dark Adapted Human Eye, Proc. Nat. Acad. Sc. **38**:80-85 (Jan.) 1952.

79. Davy, E.: The Intensity-Time Relation for Multiple Flashes of Light in the Peripheral Retina, J. Optic. Soc. America **42**:937-941 (Dec.) 1952.

80. Brindley, G. S.: Bunsen-Roscoe Law for the Human Eye at Very Short Durations, J. Physiol. **118**:135-139 (Sept. 26) 1952.

81. Ferguson, W. J. W., and Sharpley, F. W.: Dark Adaptation and Miners' Nystagmus, Commission international de l'eclairage 12th Session, 1951, Vol. 2, Paper Ee, 1952.

82. Lange, F.: Untersuchungen zur normalen Verteilung des Lichtsinns in der Netzhaut und ihrer Altersabhängigkeit, von Graefes Arch. Ophth. **153**:93-104 (Sept.) 1952.

cluded that individual variations were no greater for this (perimetric light sensitivity) than those of other clinical methods of investigation. He found also that there was a significant loss of light sensitivity over the entire retina with increasing age. A special perimeter for studying regional differences in retinal sensitivity to light stimuli, both stationary and moving, is described by Cibis, Brogan, and Fleck.[83] The effect of exposure to red light prior to dark adaptation on the degree of dark adaptation has again been investigated by McLaughlin.[84] The average dark adaptation threshold for 10 subjects was 0.151 log micromicrolambert lower after a 10-minute exposure to red light and a 10-minute period in total darkness than when they were subjected to a 20-minute period in total darkness. The conclusion is that at least a part of the process of dark adaptation proceeds more rapidly in the presence of red light than in darkness.

That local adaptation may be the explanation of the temporary loss of the ability of some pilots to see the horizon on dark nights has been given by Cibis.[85] These pilots found that by flying upside down they were able to regain perception of the horizon.

Using rigorous psychophysical methods, de Groot, Dodge, and Smith[86] have measured the light thresholds of the completely dark-adapted eye between 1 and 27 degrees from the point of fixation into the periphery in four positions: up, down, right, and left. The general light sensitivity was found to increase rapidly to a maximum between 7 and 10 degrees from the fovea and then to decrease uniformly toward the periphery. The nasal quadrant showed the best sensitivity, but the general pattern of sensitivity was the same in all quadrants except that which included the blind spot.

CONTRAST THRESHOLDS

Whereas many determinations have been made on the contrast threshold, or visibility of spots the luminance of which was higher than that of the background ($\Delta B/B$ positive), few have been made for which the spot was of lower intensity ($\Delta B/B$ negative). Bouman and Blokhuis[87] have recently made such determinations under controlled conditions for a retinal area 7 degrees from the fixation point. Circular, square, and rectangular shapes were flashed with various short exposures. The threshold was taken as that contrast which could be perceived 60% of the times the spot was exposed. The results were in accord with positive con-

83. Cibis, P. A.; Brogan, F. A., and Fleck, H. G.: Time Threshold Perimetry: A Revolving Perimeter for Studying the Regional Sensitivity and Time Factors in Visual Sensation, Project No. 21-31-006, Report No. 1, U. S. Air Force School of Aviation Medicine, Randolph Field, Texas April, 1952.

84. McLaughlin, S. C., Jr.: A Facilitative Effect of Red Light on Dark Adaptation, Project No. NM 001 059.28.01, U. S. Naval School of Aviation Medicine, Pensacola, Fla., May, 1952.

85. Cibis, P. A.: Retinal Adaptation in Night Flying, Project No. 21-31-014, U. S. Air Force School of Aviation Medicine, Randolph Field, Texas, May, 1952.

86. de Groot, S. G.; Dodge, J. M., and Smith, J. A.: Factors in Night Vision Sensitivity: Effect of Brightness, Medical Research Laboratory, Report No. 194, U. S. Naval Submarine Base, New London, Conn., March 18, 1952, Vol. 11, No. 11.

87. Bouman, M. A., and Blokhuis, E. W. M.: Visibility of Black Objects Against an Illuminated Background, J. Optic. Soc. America **42**:525-528 (Aug.) 1952.

trasts and the quantum theory of absolute threshold levels. The thresholds which are independent of the shape depend on the absolute threshold and on visual angle (square root of area), flash time, and behavior of contrast thresholds. In another study of contrast thresholds, for a spot 7 degrees in the retinal nasal periphery, in which the adapting light was green (or red) and the test spot was red (or green), Bouman [88] found, among other things, that the state of adaptation of the one eye and the perception of light in this eye were not affected or influenced by the actual stimulation or state of adaptation of the opposite eye. Influences due to pupil size must be eliminated, however. Rios Sasiain [89] had previously reported a marked influence of the adaptation level of one eye on the differential sensitivity of light of the other.

Flamant [90] studied the least perceptible difference in intensity between two point-light sources when close together and when separated for the fovea and for the peripheral retina. The differential threshold curve found was compared with the variation of visual acuity and the density of cones toward the periphery.

A historical review of the so-called metacontrast phenomenon has been made by Alpern.[91] In this phenomenon the brightness of a flashed source of light appears decreased when just preceded by another flash of light in the same neighborhood.

CRITICAL FLICKER FREQUENCY

In a rather long article, Simonson and Brožek [92] have reviewed the literature (183 references) on flicker fusion frequency, pointing out the fundamental variables, the different procedures and techniques, and the principal results of studies made on physiological stresses. Their view is that critical flicker frequency is a function of the central nervous system rather than of the retina. Peckham and Arner [93] state that the critical flicker frequency can be used to develop a "logarithmic index" of retinal sensitivity and that this is a better index than one derived from measurements of visual acuity. Yamagishi [94] also states that clinically the flicker test gives an indication of the essential sensitivity of the retina, since it is unaltered by refractive errors. The critical flicker frequency was studied by Winchell and Simonson [95] to determine the effect of the ratio of the light to the dark phases of the flicker, the light phase consisting of from 20 to 98% of each cycle. They found that the critical flicker frequency decreased nearly linearly with the logarithm of the light-dark ratio.

88. Bouman, M. A.: Peripheral Contrast Thresholds for Various and Different Wavelengths for Adapting Field and Test Stimulus, J. Optic. Soc. America **42**:820-831 (Nov.) 1952.

89. Rios Sasiain, M.: Sobre el umbral luminoso differencial de la retina, Arch. Soc. oftal. hispano-am. **11**:265-271 (March) 1951.

90. Flamant, F.: Differential Intensity Thresholds of Two Luminous Points, Rev. opt. (Théor. Instrum.) **31**:113-120, 1952.

91. Alpern, M.: Metacontrast: Historical Introduction, Am. J. Optom. **29**:631-646 (Dec.) 1952.

92. Simonson, E., and Brožek, J.: Flicker Fusion Frequency: Background and Applications, Physiol. Rev. **32**:349-378 (July) 1952.

93. Peckham, R. H., and Arner, W. J.: Visual Acuity, Contrast, and Flicker, as Measures of Retinal Sensitivity, J. Optic. Soc. America **42**:621-625 (Sept.) 1952.

94. Yamagishi, R.: A Clinical Use of Flicker Test, Acta Soc. Ophth. (Japan) **56**:820-832 (Aug.) 1952.

95. Winchell, P., and Simonson, E.: Effect of Light: Dark Ratio on the Fusion Frequency of Flicker, J. Appl. Physiol. **4**:188-192 (Sept.) 1951.

The data suggest that the critical flicker frequency depends on the refractory period of the retinal elements which occurs during the dark intervals of the flicker.

In comparing the critical flicker frequency observed binocularly and monocularly, in a repetition of Sherrington's experiments, Baker [96] concluded (as before with Bott) that binocular critical flicker frequency involves, in part, "some central process which combines and integrates the neural processes arising from stimulation of corresponding retinal areas." In line with this are the results of the experiments with the latency and duration of after-images impressed monocularly and binocularly, as reported by Misiak and Lozito.[97] In these studies the latency of the binocular after-images was significantly shorter (15%) and the duration longer (24%) than the corresponding periods of the monocular images. These results also suggest the existence of an interaction between the mechanisms of the two eyes.

Fabricant and Rose [98] state that smoking increases the critical flicker frequency, as a result of vasoconstriction. Landis and Zubin [99] found that the ingestion of phenobarbital caused a statistically significant decrease in the critical flicker frequency.

COLOR

In 1951 Hurvich and Jameson [100] reported that when they fused binocularly monocular psychologically pure red and green stimuli, their sensation was one of "white," not yellow, as Hecht and others have found. This is a crucial experiment that has considerable implication for color vision theory, and for the Young-Helmholtz three-component theory in particular. Talbot [101] criticizes these data as not being proof, on the grounds that the colors had not first been mixed monocularly to determine the color of their monocular fusion, that the criterion of pure "red" and pure "green" was not clear, and that the test field was too small—the parafoveal region is more sensitive to yellow than is the fovea. Walls [102] also discusses this experiment at great length and insists that even if the Hurvich and Jameson experiment were valid it still does not shake the three-color theory—at least his own interpretation of that theory. Walls does not grant that the experiment was valid, however, because the red and green stimuli used were complementary and their mixture could not have been seen as yellow, even monocularly. He also questions whether both observers could have had normal color vision.

Shikano reports that whereas the central part of the retina responds equally to a simple yellow light and to one obtained by mixing a lithium-red and a thallium-green light, the peripheral parts do not. This difference is found in the dark adapta-

96. Baker, C. H.: Dependence of Binocular Fusion on Timing of Peripheral Stimuli and on Central Process, Symmetrical Flicker, Canad. J. Psychol. **6**:1-10 (March) 1952.

97. Misiak, H., and Lozito, C. C.: Latency and Duration of Monocular and Binocular After-Images, J. Exper. Psychol. **42**:247-249 (Oct.) 1951.

98. Fabricant, N. D., and Rose, I. W.: Effect of Smoking Cigarettes on the Flicker Fusion Threshold of Normal Persons, Eye, Ear, Nose & Throat Month. **30**:541-543 (Oct.) 1951.

99. Landis, C., and Zubin, J.: Effect of Thonzylamine Hydrochloride and Phenobarbital Sodium on Certain Psychological Functions, J. Psychol. **31**:181-200 (April) 1951.

100. Hurvich, L. M., and Jameson, D.: Binocular Fusion of Yellow in Relation to Color Theories, Science **114**:199-202 (Aug. 24) 1951.

101. Talbot, S. A.: Green Vision and Binocular Fusion of Yellow, Science **115**:220 (Feb. 22) 1952.

102. Walls, G. L.: Binocular White from Red and Green, and the 3-Component Theory, Brit. J. Ophth. **36**:67-74 (Feb.) 1952.

tion [103] to yellow and in the size of the visual fields for yellow.[104] This result is taken to suggest the presence of yellow receptors in the retina.

In the peripheral parts of the retina, where the eye is said to be totally color-blind, Engelhardt [105] has determined the relative light sensitivity. He used a perimeter and an 18 mm. test spot and compared brightness of colors with 10 different steps of gray. All colors were seen as gray; the yellow-orange appeared the brightest, the reds and greens of medium brightness, and the blue-purple the darkest. He also studied [106] the color perception of blue in the red-green blind part of the retina. Colors containing blue were perceived as blue or violet. Some bluish-purple colors can be perceived as such that ordinarily cannot be seen as color at the center of the retina.

The appearance of colors under twilight illuminances was studied by Middleton and Mayo.[107] Squares subtending visual angles of 2 degrees were fixated centrally and binocularly. At very low levels of illumination all colors are seen as achromatic. As the illuminance is raised, purple is the color first reported, and this applied to the purple, red, and orange regions in photopic illuminations. Color vision begins at an illuminance level corresponding to full moonlight. They did not find the "blueness" frequently reported for twilight. These authors state that the tritanopia often attributed to a small region within the central fovea was here found in the 2-degree test field when the luminance was low. However, Hunt [108] did find that the color of objects under dim light and observed with the dark-adapted eye is a pale blue, and not colorless. Thomson and Trezona [109] studied the discrimination of color of the central fovea at low levels of illuminance and reported a uniform deterioration for the whole spectrum except in the region of 420 to 460 mμ, and they stated that the eye was not tritanopic at low illuminances. Weale,[110] however, reported that at low luminances the retina at 10 and at 15 degrees extrafoveally is tritanopic.

Brown [111] found that the precision of color matching was increased the larger the matching fields, and that the sensitivity for fields above 12 degrees was independent of the surround. For small matching fields the color discrimination was

103. Shikano, S.: Dark Adaptation for Yellow, Acta Soc. Ophth. (Japan) **56**:186-189 (March) 1952.

104. Shikano, S.: A Study of the Yellow-Field, Acta Soc. Ophth. (Japan) **56**:60-63 (Jan.) 1952.

105. Engelhardt, A.: Die Helligkeitsempfindungen in der total farbenblinden Zone der Netzhaut, Ztschr. Biol. **105**:232-236, 1952.

106. Engelhardt, A.: Die in der rot-grün-blinden Zone der Netzhaut blau und violett aussehenden Farben, Ztschr. Biol. **105**:83-90, 1952.

107. Middleton, W. E. K., and Mayo, E. G.: Appearance of Colors in Twilight, J. Optic. Soc. America **42**:116-121 (Feb.) 1952.

108. Hunt, R. W. G.: Light and Dark Adaptation and the Perception of Color, J. Optic. Soc. America **42**:190-199 (March) 1952.

109. Thomson, L. C., and Trezona, P. W.: Hue Discrimination of the Central Fovea Measured at Various Luminance (Brightness) Levels, J. Physiol. **112**:39P (July) 1951.

110. Weale, R. A.: Hue-Discrimination in Para-Central Parts of the Human Retina Measured at Different Luminance Levels, J. Physiol. **113**:115-122 (March 19) 1951.

111. Brown, W. R. J.: Effect of Field Size and Chromatic Surroundings on Color Discrimination, J. Optic. Soc. America **42**:837-844 (Nov.) 1952.

reduced by surrounding fields of different color. Rouse [112] found that with small fields centrally fixated, the monocular differential thresholds for colored test lights were independent of the exposure of the test lights from 0.002 to 1.0 second.

It is of interest to learn that the Purkinje shift (the wave length of maximal sensitivity of the eye shifts toward the blue when the eye adapts from high to low luminances) has been found in the fruit fly (Drosophila melanogaster) by Fingerman and Brown.[113]

When critical color matches are to be made, Winch, Harrison, and Ruff [114] found that a combination of fluorescent and tungsten lamps provides a closer approximation to daylight than do fluorescent lamps alone. Similar studies were made by Burnham, Evans, and Newhall,[115] who reported a systematic shift in the appearance of colors toward the blue when the adaptation of the eye was changed from daylight to tungsten illumination, or toward the yellow when adaptation was changed from tungsten to daylight illumination. These changes were of the order of 20 just noticeable differences.

COLOR VISION TESTING

In using pseudoisochromatic tests for color vision, Schmidt [116] found that the quality and intensity of the illumination were important. Variations in this illumination affected the behavior of persons with defective color vision, especially deuteranomalous subjects. Schmidt recommended that the pseudoisochromatic plates be used only with artificial daylight lamps, and in particular with the Macbeth daylight lamp.

The Hardy-Rand-Rittler polychromatic plates for testing color vision were evaluated in comparison with other tests, particularly with the Nagel anomaloscope, by Schmidt.[117] The results with the use of these plates proved similar to those obtained in the American Optical Company 19-plate test in that all persons with defective color vision were detected and the same number of normals were failed. The results also agree well with those obtained with the Nagel anomaloscope. The plates are widely applicable, convenient to administer, and worthy of further development. Heinsius [118] has developed a new color lantern based on existing lanterns for the detection of the person with deficient color vision. This lantern seems to offer no more than does the lantern recently developed by the Navy at the Submarine Base,

112. Rouse, R. O.: Color and the Intensity-Time Relation, J. Optic. Soc. America **42**:626-630 (Sept.) 1952.

113. Fingerman, M., and Brown, F. A., Jr.: A "Purkinje Shift" in Insect Vision, Science **116**:171-172 (Aug. 15) 1952.

114. Winch, G. T.; Harrison, W., and Ruff, H. R.: Fluorescent Lamp Artificial Daylight Units for Colour Matching, Transactions of the Illuminating Engineering Society, London, 1951, Vol. 16, No. 1, pp. 1-12.

115. Burnham, R. W.; Evans, R. M., and Newhall, S. M.: Influence on Color Perception of Adaptation to Illumination, J. Optic. Soc. America **42**:597-605 (Sept.) 1952.

116. Schmidt, I.: Effect of Illumination in Testing Color Vision with Pseudo-Isochromatic Plates, J. Optic. Soc. America **42**:951-955 (Dec.) 1952.

117. Schmidt, I.: Comparative Evaluation of the Hardy-Rand-Rittler Polychromatic Plates for Testing Color Vision, Project No. 21-31-013, U. S. Air Force School of Aviation Medicine, Randolph Field, Texas, June, 1952.

118. Heinsius, E.: Über eine neue Farblaterne zur Untersuchung von Farbenfehlsichtigkeit, Klin. Monatsbl. Augenh. **120**:86-89, 1952.

New London, Conn. Walls and Matthews [119] suggest that Maxwell's spot be used as a means of studying normal foveal color vision and color blindness. When a purple filter (Wratten 2B89) and a neutral filter (Wratten 96) are placed alternately in front of an eye of an observer who looks intently at a bright area, the observer will see an after-image-like ring surrounding the fixation point. This ring, according to Walls and Matthews, is not due to the yellow pigment surrounding the fovea, but indicates the pattern and type of distribution of the retinal receptors. By observations on the nature of this ring, conclusions can be made as to the subject's type of color vision.

COLOR VISION DEFICIENCY

Jaeger and Kroker [120] studied 16 dichromats whose color vision could always be correctly determined with the anomaloscope. These were then tested with the color wheel in matching a blue-green and a gray-purple to a gray, first with a size of visual field corresponding to 1¼ degrees (fovea) and, second, to a visual field of 22 degrees. Eight of the dichromats acted as trichromats, and these eight subjects were accordingly classified as having incomplete dichromasy. Nevertheless, these results show that the size of the visual color test field appears to be a factor in the correct classification of color-anomalous subjects. Janouskova [121] reports that the incidence of defective color vision increases with age for both sexes, especially in the beginning of the sixth decade of life. Boehmig [122] tested the effect of exertion on color perception. After the finish of a race, the color vision of the cyclists was tested for very small colored objects. He reports that a great number showed transient central color scotomas, especially for green. The scotomas were worse the greater the exhaustion of the cyclist. The author attributed the color scotomas to a transient toxic neuritis caused by chemical products of exertion.

The general characteristics of tritanopia (blue-defectiveness) were described by Wright [123] in the Adolph Lomb Memorial Lecture before the Optical Society of America in March, 1952. As a result of the publication of particular color charts in the English picture weekly *Picture Post*, 900 readers replied to the crude "mass testing." Of this number, Wright found some 17 persons with tritanopia. These persons then served as subjects for the study. The incidence of tritanopia was estimated to be from 1 in 13,000 to 1 in 65,000 persons. The ratio of men to women was of the order of 1.6:1.0. The results of measurements support the theory that tritanopia is due to the absence of retinal receptors for blue. The exact location of the fundamental stimulus associated with the blue process on the

119. Walls, G. L., and Matthews, R. W.: New Means of Studying Color Blindness and Normal Foveal Color Vision, with Some Results and Their Genetical Implications, Los Angeles, University of California Press, 1952.

120. Jaeger, W., and Kroker, K.: Über das Verhalten der Protanopen und Deuteranopen bei grossen Reizflächen, Klin. Monatsbl. Augenh. **121**:445-449, 1952.

121. Janouskova, Z.: Problém barvocitu s hlediska populacně biologického (Zhodnocení výsledku z části celkove prace), Československ. ofth. **7**:182-186, 1951.

122. Boehmig, A.: Über das zentrale Farbenunterscheidungsvermögen nach körperlichen Anstrengungen, Deutsche Gesundheitswes. **6**:1386-1388 (Nov. 29) 1951.

123. Wright, W. D.: The Characteristics of Tritanopia, J. Optic. Soc. America **42**:509-521 (Aug.) 1952.

chromaticity diagram based on the loci of colors which are confused by the tritanope has not yet been determined with precision. Fischer, Bouman, and ten Doesschate [124] made a detailed study on one person who had congenital tritanopia. It would appear that the inheritance of tritanopia is very different from that of protanopia and deuteranopia. The spectral luminosity curve of this subject differed somewhat from that usually considered characteristic for the tritanope; yet those differences occur also in variations between persons with normal color vision.

STEREOPSIS

That streoscopic depth perception is a sensation resting on its own physiological basis and is to a great extent independent of other visual functions is the thesis presented by Burian.[125] As such, therefore, stereopsis should not be considered as "the third degree of fusion." Although there is some confusion in the literature, stereopsis can indeed occur even when the disparities between the images in the two eyes are so large that the images are seen double (not fused). That this is true has recently been reaffirmed by Ogle.[126] He showed, however, that there are limits in the disparity within which stereopsis can occur with double images. With special apparatus he has measured these limiting disparities. At the macula these are about 20 minutes of arc, but they increase rapidly toward the periphery, so that at 8 degrees peripherally these limiting disparities are of several degrees. In terms of the space corresponding to these limiting disparities, there are regions beyond and nearer than the fixation point in which there is no stereoscopic depth perception. In these the double images are indefinitely localized. With distant fixation, however, stereoscopic depth occurs in the greater part of the binocular visual field. The fact that these data do show that stereopsis can exist with double images is evidence, but not proof, that the fusion function is not a factor underlying stereopsis. The existence of limiting disparities for stereopsis implies neuroanatomical limitations and a physiological basis for the phenomenon. These data obtained under controlled conditions point out the danger of one's drawing conclusions from observations such as those made with a stereoscope with large hand-drawn targets of two eccentric circles, for Scullica [127] concluded from such observations that there was practically no limit of disparity for which stereopsis could not occur.

Hamburger [128] suggests that in some persons poor stereopsis is due to horror fusionis, which ordinarily would not be detected, or to extreme ocular dominance, which he considered would cause a suppression of the binocular spatial image.

124. Fischer, F. P.; Bouman, M. A., and ten Doesschate, J.: A Case of Tritanopy, Docum. Ophth. **5–6**: 73-87, 1951.

125. Burian, H. M.: Stereopsis, Docum. ophth. **5–6**:169-183, 1951.

126. Ogle, K. N.: Disparity Limits of Stereopsis, A. M. A. Arch. Ophth. **48**:50-60 (July) 1952; On the Limits of Stereoscopic Vision, J. Exper. Psychol. **44**:253-259 (Oct.) 1952.

127. Scullica, F.: Sui limiti della fusione stereoscopica, Ann. ottal. e clin. ocul. **77**:303-322 (Aug.) 1951.

128. Hamburger, F. A.: Die Bedeutung des binocularen Wettstreites für die Stereoskopie und für die stereoskopische Entfernungsmessung, von Graefes Arch. Ophth. **153**:57-82 (Sept.) 1952.

Although confusing the limits of fusion (Panum's areas) with those of stereopsis, Walls [129] suggests that this aspect of vision might be susceptible to training—the extending of these limits and thus extending the range of useful stereoscopic depth localization. Practice, however, decreases the region of binocular single vision. Wohlzogen,[130] by means of after-images, showed that the sensation of stereopsis requires a simultaneous stimulation or after-image excitation of the two retinas. If the after-image of one eye is caused to fade by pressure on the globe, stereopsis ceases. His results also suggest that the stereoscopic effect which originates from after-images is retinal, and not cortical.

Stereoscopic depth perception under photopic and scotopic illuminations has been studied by Gumbel [131] in order to find the relative value of cone and rod vision in depth perception. He used a conventional Howard two-peg test, at a peripheral angle of 4 to 6 degrees from a red-light fixation point. For low illumination levels radium plaques were used. The actual data showing the stereoacuity in minutes of arc are not given, and the reader is left with the author's statement that at scotopic levels (rod vision) of illumination the binocular depth perception in the dark-adapted eye exists only in a rudimentary form.

It might be expected that if a subject maintains a small cyclotropia (cyclotorsion) he would have a faulty stereoscopic depth perception. The evidence is, however, that normally the eyes make cyclofusional movements in accordance with the demands of the objects being observed in the interest of single binocular vision.[132] Cibis [133] has tested 100 persons for a cyclotorsion by using a stereoscopic vision test. He found that 60% of the subjects responded most readily to changes in cyclotorsion of the eyes as demanded by the conditions of the experiment. Between 30 and 40% of the subjects failed so to respond. They gave constant depth judgments, which indicated more or less fixed cyclotorsional position of the eyes. Those of these subjects whose depth perception data were faulty were assumed to have anomalous cycloincongruities in the organization of corresponding points in the two eyes. Neither anoxia nor alcohol affects the depth judgments of these subjects. Adamson [134] investigated with the haploscope the actual apparent rotated position of stereoscopically presented horizontal arrows of unequal length in the two eyes. He found, as would be expected, that the rotation was less than that calculated from the difference in lengths of the two arrows, and that ocular scanning of the stereoscopically perceived arrow enhanced its being correctly interpreted. Empirical factors (Ogle [132]) and limiting stereopsis (Miles [135]) could in part account for his results.

129. Walls, G. L.: The Common-Sense Horopter, Am. J. Optom. **29**:460-477 (Sept.) 1952.

130. Wohlzogen, F. X.: Die Entstehung sterischer Nachbilder, Experientia **8**:194 (May) 1952.

131. Gumbel, E. J.: A Study of Depth Perception in the Light and Dark Adapted Eye, Am. J. Optom. **29**:613-623 (Dec.) 1952.

132. Ogle, K. N.: Researches in Binocular Vision, Philadelphia, W. B. Saunders Company, 1950.

133. Cibis, P. A.: Faulty Depth Perception Caused by Cyclotorsion, A. M. A. Arch. Ophth. **47**:31-42 (Jan.) 1952.

134. Adamson, J.: Ocular Scanning and Depth Perception, Nature, London **168**:345-346 (Aug. 25) 1951.

135. Miles, P. W.: Limits of Stereopsis Due to Physiologic Diplopia, Am. J. Ophth. **32**:1567-1573 (Nov.) 1949.

The techniques that can be used, and that he has used so successfully for making stereoscopic drawings, are described by Allen.[136] Anyone who has seen his stereoscopic drawings, for example, those showing the relationships of nerves and vessels to the brain stem, and also the drawings showing the course of the optic nerve tract and radiations, will be much impressed with their excellence and their value for teaching. Asher and Law [137] describe a new stereoscope for clinical work that eliminates the many objectionable features of those currently available, in that the accommodation demand and convergence can be controlled separately or in any given relationship.

HOROPTER

In a mathematical discussion of the horopter problem, Fischer, Ludwig, and Wartmann [138] start with the hypothesis that there is a fixed innate arrangement of corresponding points of the two retinas resulting in a curvature of the horopter that changes from an in-curve for a near convergence to an out-curve for a more distant convergence. The fixed arrangement causes an undistorted (flat) horopter at some distance h_0. With a given convergence, then, all space beyond and nearer the horopter will be distorted. However, for minimal distortion in the whole binocular space, the distance h_0 should be equal to twice the distance corresponding to the near point of convergence, namely, 40 to 50 cm. The authors analyze the data of four independent observers which are to be found in the literature and show that for these four investigators h_0 lies between 35.9 and 48.0 cm. Thus, the authors consider the point proved. I feel that the fundamental premise is incorrect, for existent data show that the arrangement between corresponding points apparently changes with convergence.

The influence of heterophoria on the position of the region of binocular single vision relative to the point of fixation has been investigated by Vukovich and Müller.[139] By obtaining the disparities between the images of the two eyes corresponding to the "doubling" point, they reaffirmed that there was a shift depending on whether the observer was esophoric (nearer) or exophoric (behind). This shift is, of course, the fixation disparity.

PULFRICH STEREOPHENOMENON

The Pulfrich stereophenomenon (the path of the bob of a swinging pendulum appears to be elliptical when one eye is darkened with a filter) has been explained by Fischer and colleagues as essentially a phenomenon of optokinetic eye movements instead of a difference in the time of perception of the two eyes. Dittler, Rosemann, and Buchmann [140] object to this explanation on the grounds that changes in convergence should cause a change in the apparent distances of all

136. Allen, L.: Stereoscopic Drawing Techniques, M. & Biol. Illus. **1**:182-200 (Oct.) 1951.

137. Asher, H., and Law, F. W.: Stereoscopy and a New Stereoscope, Brit. J. Ophth. **36**:225-239 (May) 1952.

138. Fischer, F. P.; Ludwig, W., and Wartmann, R.: Der optimale Horopter: Mit einer Konsequenz zur Erklärung der subjektiven Himmelskrümmung, Arch. ges. Physiol. **254**:377-392 (March 15) 1952.

139. Vukovich, V., and Müller, J.: Heterophorie und Binocularsehen, von Graefes Arch. Ophth. **153**:83-92 (Sept.) 1952.

140. Dittler, R.; Rosemann, H. U., and Buchmann, H. H.: Zur Deutung des Pulfrich-Effektes: Mitteilung, Ztschr. Biol. **105**:40-48, 1952.

stationary objects in the field of view, and that such a theory would necessitate a change in distance of real objects with each change of convergence. Fischer,[141] however, maintains his thesis. With the continuous movements of the eye, the visual field is continuously fluctuating, together with its objects of vision. Rosemann and Buchmann [142] then recorded eye movements using the corneoretinal potential method during the time that the Pulfrich effect was being observed. When the midpoint of the swing of the pendulum was fixated and the stereoscopic effect was strong, no regular optokinetic movements were evident in the tracings, and no movements greater than 1 or 2 degrees occurred. Thus the swinging pendulum did not disturb the equilibrium of the stabilizing factors of the field of view, and optokinetic movements could not be the basis of the phenomenon.

SPACE PERCEPTION

Rose [143] pointed out that it is not possible to predict the quality of spatial orientation by a test for a single depth perception factor or clue. For pilots he expressed the belief that monocular clues (for example, familiar size) and motion parallax are the limiting factors, and that for pilot selection only these two factors need be considered. However, stereoscopic factors cannot be ruled out, for the acuity for depth perception by motion parallax is of the same order as that for stereopsis. Hochberg and Hochberg [144] performed experiments to determine the extent to which past experience is essential to depth perception. Reversible-screen (panel) drawings upon which figures of a boy and man of equal size were drawn, and a second drawing of the same boy of different sizes, were used as test objects. The extent to which there was familiarity with "boy" and "man," and therefore a dominance of one or the other panel, should cause the corresponding drawing to appear nearer. They found that familiar size was not a factor in space perception under these conditions, although relative size was. The latter, in their opinion, does not require "preperceptual recognition of retinal images, past experience, and unconscious inference."

The vertical-horizontal illusion in which the vertical dimension of lines appears longer than does the horizontal dimension of lines of equal length was studied by Sleight and Austin [145] to learn whether the illusion also held for plane figures. For the circle-ellipse figures it did, but for the square-rectangle figures in half the number of subjects it did not. Osgood and Heyer [146] have shown that the figural after-effect phenomenon (wherein the fixation of a given figure for a period of time influences the apparent size or form of another figure subsequently fixated) can

141. Fischer, M. H.: Betrachtungen über den optischen Raumsinn, Ztschr. Biol. **105**:66-72, 1952.

142. Rosemann, H. U., and Buchmann, H. H.: Zur Deutung des Pulfrich-Effektes, Ztschr. Biol. **105**:134-140, 1952.

143. Rose, H. W.: Monocular Depth Perception in Flying, J. Aviation Med. **23**:242-245 (June) 1952.

144. Hochberg, C., and Hochberg, J.: Familiar Size and the Perception of Depth, J. Psychol. **34**:107-114 (July) 1952.

145. Sleight, R. B., and Austin, T. R.: The Horizontal-Vertical Illusion in Plane Geometric Figures, J. Psychol. **33**:279-287 (April) 1952.

146. Osgood, C. E., and Heyer, A. W., Jr.: A New Interpretation of Figural After-Effects, Psychol. Rev. **59**:98-118 (March) 1952.

be explained on the "neurophysiological principles concerning a nervous system composed of single neurones with precise connections," as developed by Marshall and Talbot, as against the electrical field theory postulated by Köhler and Wallach.

The discovery of muscle spindles in the eye muscles has reopened the problem of a proprioceptive sense from the muscles. Cooper, Daniel, and Whitteridge [147] studied the action potentials of sheep and goat muscles, and they report that a stretch in the muscle produced a rapid acceleration of these impulses, which decreased when the muscle was released. The nerve endings in the muscles send afferent discharges up the oculomotor nerve. Because of the similarity to the eye muscles in man, these authors infer that a proprioceptive sensitivity of those muscles can play a part in control of eye movements, even for movements of the order of 1.5 degrees. Ludvigh,[148] however, presented data from experiments which indicate that any position sense from the extrinsic muscles of the eyes is very poor, at least for movements less than 6 degrees. Ludvigh [149] suggests that the chief function of the muscle spindles in the extrinsic muscles is to provide a "feed-back," which subsequently alters the response of a given muscle to a motor stimulation, such as would be involved, for example, in pursuit movements. Only the distance of an image from the fovea can provide the clue (the innervation for an eye movement) on which the subject can base his sense (correct or incorrect) of his eye position.

PHYSIOLOGICAL NYSTAGMUS

The phenomenon of the continuing and fluctuating small involuntary eye movements (physiological nystagmus) has become of special interest the past several years. Whether the phenomenon actually exists, how large are the excursions, and what are their distributions have led to some disagreement. Walls [150] has reviewed the literature and has shown that the larger of these nystagmoid movements (synchronous for the two eyes) can be observed simply in certain surroundings, such as the lights of a Christmas tree if one lamp is fixated continuously. However, it is the smaller unsynchronized movements that are being investigated. By photographing a blood vessel of the eye, Higgins and Stultz [151] were able to record the motions of the eye during steady fixation, in both horizontal and vertical directions, monocularly and binocularly. The very rapid motions have a frequency of about 50 per second and an average amplitude of 1.2 minutes of arc. Riggs [152] has recently modified the method developed by Lord and Wright in which the light of a ribbon tungsten lamp reflected from the cornea forms an image of the ribbon on a straight edge. The amount of light passing the straight edge is reg-

147. Cooper, S.: Daniel, P. M., and Whitteridge, D.: Afferent Impulses in the Oculomotor Nerve, from the Extrinsic Eye Muscles, J. Physiol. **113**:463-474 (May 28) 1951.

148. Ludvigh, E.: Possible Role of Proprioception in the Extraocular Muscles, A. M. A. Arch. Ophth. **48**:436-441 (Oct.) 1952.

149. Ludvigh, E.: Control of Ocular Movements and Visual Interpretation of Environment, A. M. A. Arch. Ophth. **48**:442-448 (Oct.) 1952.

150. Walls, G. L.: An Autodemonstration of the "Physiologic Nystagmus," Am. J. Ophth. **35**:231-235 (Feb.) 1952.

151. Higgins, G. C., and Stultz, K. F.: Frequency and Amplitude of Ocular Tremor, abstracted, J. Optic. Soc. America **42**:872 (Nov.) 1952.

152. Riggs, L. A.: Measurement of Normal Ocular Tremor by Corneal Reflection, J. Optic. Soc. America **42**:287 (April) 1952.

istered by a photomultiplier tube. Eye movements result in changes in the registration. The records of the small ocular tremors of the order of 30 to 90 vibrations per second with rotations of 15 to 20 seconds of arc found by this method are similar to those he found previously by photographing the image from a contact lens.

One theory about the physiological nystagmoid movements is that they tend to improve visual acuity by providing temporal variation of the stimulations of neighboring retinal elements. Ratliff,[153] however, has shown that for grating test objects the involuntary drifts of the visual axis and the larger minute rapid tremors are clearly detrimental to monocular visual acuity. In a very ingenious experiment Riggs and Ratliff[154] provided a fixation mark, projected on a screen, resulting from the reflection of a small light beam from a corneal contact lens, arranged in such a way that any motion of the eye causes a deviation of the fixation image on the screen which exactly offsets the deviation the image would have on the retina. Thus, regardless of the eye movement, the retinal image of the fixation point remains on the same retinal receptor. With this arrangement the authors found, in comparison with normal viewing, that when the target is first presented the visual acuity is better than normal but within a few seconds the lines of the grating disappear and within one minute even coarse lines are seen only intermittently. The conclusion is that normal eye movements are important because they prevent local adaptation of the retina. In an almost identical arrangement Ditchburn and Ginsborg[155] also set up an optical system and a contact lens so that the retinal image would be "stabilized." They found that the image would disappear at intervals, that the contrast threshold was greatly increased. Small involuntary nystagmoid movements are therefore considered important for sensory perception.

CONVERGENCE

Bedrossian[156] showed that in 92% of 119 subjects with normal eyes the measurement of the diplopia exhibited when a red lens was held before one eye and the subject observed a small source of light was the same as the heterophoria measured by the Maddox rod. The image through a blue lens, on the other hand, fused with the light, as did also the images through red and green lenses. Although it is generally believed that heterophoria does not change with age, Kephart and Oliver[157] determined the correlation of lateral heterophoria and age in 6,000 industrial employees. The correlation coefficients were small (although the authors consider them significant), indicating a slight trend toward esophoria for distant vision and toward exophoria for near vision.

153. Ratliff, F.: Role of Physiological Nystagmus in Monocular Acuity, J. Exper. Psychol. **43**:163-171 (March) 1952.

154. Riggs, L. A., and Ratliff, F.: Effects of Counteracting the Normal Movements of the Eye, abstracted, J. Optic. Soc. America **42**:872-873 (Nov.) 1952.

155. Ditchburn, R. W., and Ginsborg, B. L.: Vision with a Stabilized Retinal Image, Nature, London **170**:36-37 (July 5) 1952.

156. Bedrossian, E. H.: Significance of Red-Lens Diplopia: Its Relation to Heterophoria, Am. J. Ophth. **35**:1356-1357 (Sept.) 1952.

157. Kephart, N. C., and Oliver, J. E.: A Study of the Relationship Between Lateral Phoria and Age, Am. J. Optom. **29**:423-429 (Aug.) 1952.

A study of the effect of artificial aniseikonia on the amplitudes of vertical prism divergences has been made by Ellerbrock.[158] Afocal magnification lenses of 5 or 10% were introduced before one eye of his subjects to create the artificial aniseikonia. In very careful measurements of the vertical prism vergences, he found that the effect of the aniseikonia was greatest for large target sizes (17 by 12.4 degrees), for which the amplitudes were reduced to nearly one-half those obtained without the lenses. For small target sizes (2 by 1.5 degrees) the reduction due to the lenses was small. This effect is explained on the basis that with the artificial aniseikonia of 5% the disparity between the images is such that beyond a peripheral angle of about 2 to 3 degrees the images in the two eyes fall outside Panum's areas of fusion and are accordingly imperfectly fused, if at all. Thus, the effect of the aniseikonia is to reduce the effective target size used to obtain the vertical prism vergences.

In 623 young men with no refractive errors, Krümmel[159] measured the latent lateral heterophoria by standard techniques, using interrupted and continuous observation on the part of the subjects. He found orthophoria in 12% (although in the strictest sense there was orthophoria in 3.8%), esophoria in 43%, and exophoria in 45%. The heterophoria measurements were on the whole not very stable, with tendencies to increase or decrease. The exophoria was more stable than the esophoria, a difference which is explained on the basis of the physiological tonus of the muscle synergies involved.

By means of the corneoretinal potential method of recording eye movements, Lord[160] has shown that in the act of making a voluntary convergence without a near fixation point the eyes make saccadic rotations of successive converging and relaxing movements. The average amplitude in the horizontal of these saccadic movements is about 1 degree of arc. The records for two of the subjects showed a quick divergent movement prior to the convergence.

By photographing the beam of light reflected from a small flat surface on a contact lens, Ginsborg[161] measured the eye movements that occur during blinking. His results show a simultaneous inward and upward rotation of 20 to 100 minutes of arc, in the time interval of 0.04 to 0.09 second.

The various definitions of "tonic convergence" as originating with Maddox's classification of total convergence movements are discussed by Hebbard.[162] He concludes that the tonic convergence which exists for a distant fixation point with disassociated eyes should be measured from the "true position of rest," rather than from the primary position, as in ordinary measurements of heterophoria. Since the true position of rest cannot be determined clinically, the true tonic convergence cannot be measured. Fujino[163] reports evidence for a "proximal" convergence

158. Ellerbrock, V. J.: Effect of Aniseikonia on the Amplitude of Vertical Divergence, Am. J. Optom. **29**:403-415 (Aug.) 1952.

159. Krümmel, H.: Differentielle Phorometrie, Klin. Monatsbl. Augenh. **120**:134-143, 1952.

160. Lord, M. P.: Binocular Eye Movements when Convergence Is Subjectively Changed, Nature, London **169**:1011-1013 (June 14) 1952.

161. Ginsborg, B. L.: Rotation of the Eyes During Involuntary Blinking, Nature, London **169**:412-413 (March 8) 1952.

162. Hebbard, F. W.: Measuring Tonic Convergence, Am. J. Optom. **29**:221-230 (May) 1952.

163. Fujino, S.: Studies on Convergence, Acta Soc. ophth. (Japan) **56**:142-154 (March) 1952.

when he found that, although he eliminated accommodation by plus lenses and fusion by use of separate test objects, there nevertheless was a convergence with near work.

In a long paper, Piper [164] discusses the relation of the sensory and motor functions of the eyes, with emphasis on space perception. According to him, both are involved, one or the other being the more active, depending on whether the visual surroundings involve motion or are stationary. A disturbance of either function from its habitual role results in a disturbance of spatial localization. The organism cannot tell which of the functions concerned is disturbed. According to Schubert,[165] the binocularly conjugated muscle movements are regulated by the central nervous system in the frontal cortical areas, which belong to the pyramidal system. The retinal stimulations from the optical images from objects of space pass through the occipital cortex, as a relay station, to the tonic control of the extraocular muscles. Piper [166] points out that the present point of view regarding heterophoric (and heterotropic) disturbances of abnormal motor impulses is explained by abnormal vegetative impulses, rather than on the theory of a faulty position of rest.

ACCOMMODATION

In an interesting satistical study, Ripple [167] has found that the near point of accommodation increases when the eyes are turned down or in or both. When they are turned in 40 degrees, the near point increases (statistically) from 9.8 to 11.4 D.; when they are turned from up and out to down and in, the accommodative near point increases from 8.75 to 14.1 D. Even 25% of the presbyopic subjects showed an increase. In part, these results are explained on the basis of the accommodation-convergence association. Piper [168] also discusses the findings that in exophoria the range of accommodation is increased, while in esophoria it is decreased.

LeGrand [169] is of the opinion that in the relaxed state of accommodation the punctum remotum should not be considered a static condition and rigidly fixed. The lens in this state is in an equilibrium of forces influenced by many factors, and is not in a condition of rest. In certain subjects the punctum remotum may represent the state of a positive or of a negative accommodation. Pau [170] shows that in the change from the accommodated state to the relaxed state there is an actual forward displacement of the lens as a unit (not the posterior surface).

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164. Piper, H. F.: Über die sensorische und motorische Funktion des Auges, von Graefes Arch. Ophth. **152**:425-476 (March) 1952.

165. Schubert, G.: Die Leistungen der Augenmuskeln und ihre Steuerung, Wien. klin. Wchnschr. **63**:632-634 (Sept. 7) 1951.

166. Piper, H. F.: Die Bedeutung von Augenhaltung und Akkommodation für die Klinik der vegetativen Dystonien, Med. Klin. **47**:370-372 (March) 1952.

167. Ripple, P. H.: Variation of Accommodation in Vertical Directions of Gaze, Am. J. Ophth. **35**:1630-1634 (Nov.) 1952.

168. Piper, H. F.: Augenhaltung und Akkommodationsimpuls, von Graefes Arch. Ophth. **152**:719-730 (June) 1952.

169. LeGrand, Y.: Sur les notions de "remotum" et d'accommodation négative, Année psychol. **50**:179-184, 1951.

170. Pau, H.: Die akkommodative Linsenverschiebung als Ausdruck antagonistisch wirkender Kräfte, Ophthalmologica **124**:239-253 (Oct.) 1952.

The laboratory and clinical results on accommodation-convergence association have been well reviewed by Morgan.[171] He points out that the convergence function is best interpreted in terms of the Maddox classification: tonic convergence, accommodative convergence, fusional convergence, and proximal convergence (effect of apparent nearness of object viewed). Most important is his conclusion that the accommodative convergence (the change in convergence due to an accommodative effort) is extremely stable for any one subject and appears linear with the amount of accommodative effort. Binocular vision through correcting ophthalmic lenses is made possible by the change in relative convergence, and not by a change in the accommodation-convergence association.

ELECTRORETINOGRAPHY

Judging from the number of papers on the electrophysiology of the eye, there has been a marked increase in the general interest in this field. It is not possible to give a complete review here. In human electroretinography the standard technique appears to be to fit over the cornea a contact lens into which has been fused a suitable electrode. With a second electrode on the head, the change in potential between the leads from the two electrodes can be measured by a suitable amplification system when light is introduced into the eye. With a flash of light the typical electroretinogram consists of a small negative displacement (the a-wave), followed by a rapidly positive displacement, which rises to a maximum (the b-wave) and then gradually returns to the base line. In animals there may be a c-wave, which is a second positive maximum. The electroretinographic potential, especially the b-wave, seems to be primarily a functon of the rods of the retina. This makes the method as a test of macular function of no value (Jacobson and O'Brien[172]). The results of Boynton and Riggs[173] seem to indicate that the human retinal response is aroused primarily by stray light rather than by the light focused on a particular area of the retina. The effect of change in area or intensity of the light stimulus merely increases the illumination in the nonfocal areas. The a-wave requires rather high intensities and was supposed to be a function of the cones. This wave was investigated by Armington, Johnson, and Riggs[174] and was found to consist of two components, one that develops best with red light, and therefore is thought to be a photopic (cone) response, and one that develops after prolonged dark adaptation, and therefore would be a scotopic (rod) response. Armington[175] also studied the b-wave, for Adrian had suggested that there was a brief early positive wave that just preceded the b-wave proper. This early wave has been called the x-wave. It is maximal for a wave length of about 630 mμ and is thought to be a photopic component of the b-wave. Its existence suggests there

171. Morgan, M. W., Jr.: Relationship Between Accommodation and Convergence, A. M. A. Arch. Ophth. **47**:745-759 (June) 1952.

172. Jacobson, J. H., and O'Brien, J. M.: Electroretinography: A Clinical Survey, Am. J. Ophth. **35**:1346-1348 (Sept.) 1952.

173. Boynton, R. M., and Riggs, L. A.: Effect of Stimulus Area and Intensity upon the Human Retinal Response, J. Exper. Psychol. **42**:217-226 (Oct.) 1951.

174. Armington, J. C.; Johnson, E. P., and Riggs, L. A.: The Scotopic A-Wave in the Electrical Response of the Human Retina, J. Physiol. **118**:289-298 (Nov. 28) 1952.

175. Armington, J. C.: A Component of the Human Electroretinogram Associated with Red Color Vision, J. Optic. Soc. America **42**:393-401 (June) 1952.

is a retinal mechanism responding to red light. That it is possible to inhibit the scotopic responses to a flash of light if that flash is preceded by a flash at proper interval and intensity is reported by Dodt.[176] Thus, the human retina can be made to show the "electroretinographic characteristics of a photopic retina as soon as the active inhibition proceeding from the scotopic excitation is removed from the photopic excitation processes."

Schubert and Bornschein [177] found that there was a complete lack of the b-wave in congenital night blindness and that there was a lack of x-waves in protanopes for stimulation with light of longer wave lengths than 600 mμ. This absence of the b-wave was also found by Björk and Karpe [178] and others for patients with retinitis pigmentosa, and a subnormal response was found in those with secondary pigmentary degeneration of the retina. The b-wave was also found to be subnormal in persons having retinal detachment, the magnitude depending on the area of the detachment and the vitality of the retina (Karpe and Rendahl [179]).

Monnier [180] has determined that the time required for "cortical opto-motor integration" was 75 milliseconds, obtained from the following data: The time required for a motor response (reaction time recorded electromyographically from the finger) was 120 milliseconds; the electroretinogram, the time from flash to response, was 30 milliseconds; the retinocortical time was 5 milliseconds, and the time of conduction of efferent impulses from cortical motor area to muscle was 10 milliseconds.

With an electroencephalographic type of apparatus to measure cortical currents, Köhler, Held, and O'Connell [181] placed an active electrode over the region of the cortical fovea of the head and the grounded electrode on the vertex. While the subject fixated a faint mark in the middle of the field, a bright object was made to move slowly across the visual field past the fixation point. According to the authors, all electroencephalographic records show responses to the moving object, although the particular form of the response varies. Thus, in relation to the vertex, "a positive potential develops under the occipital electrode when a bright object appears in the visual field." This result will evoke considerable skepticism.

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176. Dodt, E.: Beiträge zur Elektrophysiologie des Auges: II. Über Hemmungsvorgange in der menschlichen Retina, von Graefes Ophth. **153**:152-162 (Sept.) 1952.

177. Schubert, G., and Bornschein, H.: Beitrag zur Analyse des menschlichen Elektroretinogramms, Ophthalmologica **123**:396-413 (June) 1952.

178. Björk, A., and Karpe, G.: The Clinical Electroretinogram: V. The Electroretinogram in Retinitis Pigmentosa, Acta ophth. **29**:361-376, 1951.

179. Karpe, G., and Rendahl, I.: The Clinical Electroretinogram: VI. The Electroretinogram in Detachment of the Retina, Acta ophth. **30**:303-316, 1952.

180. Monnier, M.: Retinal, Cortical and Motor Responses to Photic Stimulation in Man: Retino-Cortical Time and Opto-Motor Integration Time, J. Neurophysiol. **15**:469-486 (Nov.) 1952.

181. Köhler, W.; Held, R., and O'Connell, D. N.: An Investigation of Cortical Currents, Proc. Am. Philosophical Soc. **96**:290-330 (June) 1952.

Annual Reviews

STRABISMUS

Review of the Literature

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IN THE preparation of this review, the attempt has been made to include the significant publications in 1952 pertaining to strabismus. From such a survey it is hoped that the reader will be given a fairly complete picture of the contemporary writings on strabismus and related subjects.

The most important event in 1952 related to the field of motility was the loss, by an untimely death, of Richard Scobee. This loss was offset, to a degree, by the publication of the second edition of his textbook, "The Oculorotary Muscles." [1] This volume contains many changes and additions, including most of the articles he had published after his first edition. It is unquestionably the best textbook in the field. I feel that it should be studied by all ophthalmologists.

ETIOLOGY

Little new material was found on the etiology of strabismus. An article by Keiner [2] merely restates the principal facts and conclusions presented in his monograph in 1951. Masters [3] found 19 pairs of twins with strabismus recorded in the literature and reported one more pair. In monozygotic twins, he concludes, the presence of a squint in one twin is generally accompanied by a similar squint in the other; the refractive errors, however may vary widely.

Stillerman, Gibbs, and Perlstein [4] studied the electroencephalograms of 1,345 children with or without organic brain disease and with or without ocular disorders. There were 1,245 children with evidence of brain disease, of whom 361 had eye pathology; 73% of these had strabismus. Of the 100 children with eye pathology alone, 89 had strabismus. Only 0.5% of 180 normal children had abnormalities in the occipital leads, while of the 89 normal children with strabismus 20% showed electroencephalographic abnormalities. In children with cerebral palsy, electroencephalographic foci in the occipital region were twice as frequent in the presence of ocular abnormalities and six times as frequent when there were seizures and ocular changes. Occipital abnormalities were twice as frequent in exotropia as in esotropia. The authors found no correlation between electroencephalographic abnormalities and fusion, the laterality of the deviation, or retinal correspondence.

1. Scobee, R. G.: The Oculorotary Muscles, Ed. 2, St. Louis, C. V. Mosby Company, 1952.

2. Keiner, G. B. J.: The Etiology of Squint, Nederl. tijdschr. geneesk. **9**:43-54, 1952; abstracted, Excerpta med. XII **6**:447, 1952.

3. Masters, S.: Strabismus in Twins, Am. J. Ophth. **35**:1824-1826, 1952.

4. Stillerman, M. L.; Gibbs, E. L., and Perlstein, M. A.: Electroencephalographic Changes in Strabismus, Am. J. Ophth. **35**:54-63 (May Pt. 2) 1952.

Holt [5] found that the incidence of divergent strabismus is higher in Australia than it is in London. She suggests that this may be due to the greater amount of sunlight in Australia.

PERIPHERAL FUSION

The writing of Burian and Swan on peripheral fusion have aroused interest in Great Britain. Stanworth [6] believes that some of Burian's observations were not truly peripheral, but were, rather, paramacular. He points out that all the measurements made on the synoptophore are confined to the paramacular region but that there is no essential differences between fusion phenomena in the periphery and those in the paramacular region; he suggests that since stimuli are more effective the nearer they are to the fixation point, one is not losing anything by using the paramacular region. Both Stanworth and Boyle [7] followed Swan's method of investigating peripheral fusion with a graduated series of rings on the synoptophore and reached essentially similar conclusions: In an infant peripheral fusion probably develops first, while the macula is still underdeveloped. In amblyopia peripheral fusion may hold the eyes straight. Peripheral fusion may play a part in the straightening of eyes in accommodative squint. The possibility of gradual encroachment on the macula from the periphery as a form of fusion training is suggested.

AMBLYOPIA

Dyer and Bierman [8] investigated the changes in cortical potential in suppression amblyopia. They found abnormal cortical brain waves by electroencephalography, which they believe support Chevasse's theory that suppression amblyopia is caused at a cortical level. They assert that suppression is associated with abnormal neuronal discharges in the cortex and suggest that electroencephalographic tracings may help in the diagnosis and prognosis of suppression amblyopia. Burian and Watson,[9] also using a standard electroencephalogram but with controlled illumination, observed the following changes: 1. If the visual acuity of the amblyopic eye was 20/70 or less, alpha activity continued when the amblyopic eye was exposed to stroboscopic light stimulation, whereas it was immediately suppressed on exposure of the normal eye. 2. The rhythmic electric response from the occiptal cortex in response to rhythmic illumination of the eyes ("photic driving") was less easily produced in some amblyopic eyes. The driving obtained by illuminating amblyopic eyes was less regular, often occurred momentarily, and was of lower voltage than that obtained by illuminating the normal, fellow, eyes.

Fixation anomalies in amblyopia have been investigated by Brock and Givner.[10] They point out the similarity of the acuity gradient and the retinal gradient at the

5. Holt, R.: A Study of Divergent Strabismus in Australia, Brit. Orthop. J. **8**:95-100, 1951.

6. Stanworth, A.: Paramacular and Peripheral Fusion, Tr. Ophth. Soc. U. Kingdom **72**:613-622, 1952.

7. Boyle, N. W.: Investigations into Peripheral Fusion, Brit. Orthop. J. **8**:50-55, 1951.

8. Dyer, D., and Bierman, E. O.: Cortical Potential Changes in Suppression Amblyopia, Am. J. Ophth. **35**:66-68, 1952.

9. Burian, H. M., and Watson, C. W.: Cerebral Electric Response in Intermittent Photic Stimulation in Amblyopia ex Anopsia: Preliminary Report, A. M. A. Arch. Ophth. **48**:137-143, 1952.

10. Brock, F. W., and Givner, I.: Fixation Anomalies in Amblyopia, A. M. A. Arch. Ophth. **48**:775-786, 1952.

fovea. Without a secondary macula, no fixed secondary retinal correspondence could be established in an amblyopic eye. With employment of the usual after-image equipment, they describe a new method capable of much more accurate measurement of fixation; with the amblyopic eye occluded, the occluder is shifted to the fixing eye. If the after-image falls to either side of the fixation dot, there is eccentric fixation. The shift can be measured accurately. They found that 20% of amblyopic eyes fixate along the central foveal axis. Most fixate eccentrically, the shift away from the center being greater the more profound the amblyopia. The best acuity of the amblyopic eye is not on the anomalous corresponding off-foveal area but is frequently on the opposite side of the true fovea. The acuity gradient in most cases of amblyopia shows a small, very circumscribed central depression of vision. This cannot be explained by obligatory cortical inhibition.

ANOMALOUS CORRESPONDENCE

Fusional phenomena in anomalous correspondence are the subject of Halldén's 90-page monograph.[11] He accepts anomalous correspondence as an adaptation phenomenon but admits that nonharmonious correspondence has caused difficulties in this explanation. The purpose of this study was to see whether fusional phenomena occur with pure anomalous correspondence and to study these phenomena. Harmonious anomalous correspondence serves a purpose; the angle of squint is compensated, and diplopia and confusion are eliminated. Nonharmonious anomalous correspondence does not serve a purpose in the same way. A condition necessary for the development of nonharmonious anomalous correspondence is that there was already lack of harmony during the time when anomalous correspondence developed. Another is that the lack of harmony occurs during free vision, and has not been brought about by artificial testing conditions. Using a combination of projected polarized targets and after-images, he disturbed free binocular vision as little as possible. He found a covariation of the objective angle and the angle of anomaly. It was a combination of fusional movements, sensory fusion, and a third component which is not fusional in character and was only indirectly indicated in his experiments. The combination of these three components resulted in the actual maintenance of harmony in spite of the variation of the objective angle. He raises the question of whether the fusional phenomena are a further development and differentiation of the adaptation which brought about the anamalous correspondence or whether they are unimportant rudiments of normal fusional movements and sensory fusion of normal occurrence.

Haldén drew the following conclusion:

> If a strabismus with harmonious anomalous correspondence is operated on, lack of harmony results. The fusion will cause an attempt to compensate for this in various ways: by a fusional movement, by sensory fusion, or by a combination of the two. The fusional movement means that the eyes try to return to the old position. It sometimes happens that the cosmetic effect of the operation is in this way nullified. By sensory fusion the angle of anomaly is adapted to the changed relative position of the eyes. It seems probable that there is often a combination of fusional movement and sensory fusion.

11. Halldén, U.: Fusional Phenomena in Anomalous Correspondence, Acta ophth., 1952 Supp. 37.

The distribution of corresponding points over the entire binocular space was studied by Kretzschmar,[12] using polarized projectors. He found that the subjective angle remains practically the same over the entire space, showing that reallocation of local signs had occurred over most of the binocular field.

Starkiewicz[13] finds that the result of the after-image test is influenced considerably by turning the patient's head. He believes that the after-image may give the earliest evidence of breakdown of normal correspondence and thus will indicate abnormal retinal correspondence when the other tests show normal correspondence.

Believing that confusion exists in the concept of abnormal retinal correspondence, Pascal[14] attempts to clarify the distinction between the two phases of normal and abnormal retinal correspondence. The two phases are (1) bimacular separation of the images (diagnosed by the after-image test) and (2) macular-comacular superposition of the images (diagnosed by comparing the objective and the subjective angle).

To determine the prognosis in abnormal retinal correspondence, Shepherd[15] included in her study only the cases with a subjective angle that was smaller than the objective angle. Using the major amblyoscope, the after-image, and double images, she found only 51 cases out of 1,104, or 4.6%, that satisfied this definition. All were cases of esotropia of low degree, rarely 20 degrees, and in most there was some amblyopia. The prognosis is good when abnormal retinal correspondence has been diagnosed before it has had a chance to become deeply rooted and occlusion can be carried out immediately, and when anomalous correspondence is found on the synoptophore only. The prognosis is fair when squint is of longer duration when abnormal retinal correspondence is found on the synoptophore and by double images, when normal correspondence is appreciated after one or two treatments, and when there is unharmonious anomalous correspondence. The prognosis is poor when the diagnosis of abnormal retinal correspondence is made with all three tests, when it persists after several treatments, and when it is harmonious. Shepherd found unharmonious anomalous correspondence more frequent, but hard to explain. It may come from a reduction of the angle after operation. She favors preoperative treatment of abnormal retinal correspondence. If normal correspondence is not achieved before operation, a small residual angle may tend to return to the preoperative angle.

METHODS OF EXAMINATION

Pointing out that the usual routine tests for convergence bring out variable amounts of voluntary and fusional convergence, Capobianco[16] suggests measuring

12. Kretzschmar, S., Jr.: Abnormal Retinal Correspondence, Ophthalmologica **123**:343-348, 1952.

13. Starkiewicz, W.: Value of Different Tests of Retinal Correspondence and the Influence of Rotation of the Head on the After-Image Test, Klin. oczna **21**:35-40, 1951; abstracted, Ophth. Lit. **5**:1187, 1952.

14. Pascal, J. I.: The 2 Phases of Abnormal Retinal Correspondence, Am. J. Ophth. **35**:1204-1206, 1952.

15. Shepherd, A. M. B.: Prognosis in Abnormal Retinal Correspondence, Brit. Orthop. J. **8**:119-123, 1951.

16. Capobianco, N. M.: Subjective Measurement of the Near-Point of Convergence and Its Significance in the Diagnosis of Convergence Insufficiency, Am. Orthop. J. **2**:40-42, 1952.

the near point of convergence in the usual way except that a red glass is held over one eye. This she calls the subjective near point, to differentiate it from the objective near point taken in the usual way, and she has found that it tends to agree with the measurement taken on the troposcope. In the diagnosis of convergence insufficiency, good amplitudes, a good objective near point, and a poor subjective near point mean trouble; good objective and subjective near points but only fair amplitudes indicate a good prognosis.

Kretzschmar [17] has adapted the Goldmann perimeter for plotting and measuring ocular deviations by using the usual red projector and adding a blue projector and red-blue glasses. It can also be adapted to the Hess method. Falkowska [18] uses a glass Hess screen and locates the deviating eye by the corneal reflex from a flashlight on the back of the screen. He has found that most pronounced concomitant squints have a paralytic element and that vertical deviations are very frequent. Overaction of the inferior oblique and inferior rectus muscles was commonest.

Thompson [19] compares the measurements made with the cover test and those made on the troposcope. The distance measurements were consistently and significantly higher on the troposcope, probably owing to instrument accommodation. Near measurements were practically identical with the two methods.

The spontaneous changes in refraction and angle of squint during the ages of growth were studied by Nordlöw [20] in 122 children. His routine included refraction under atropine, acceptance when possible, estimation of the angle by the Hirschberg method, measurement on the perimeter, retinal correspondence by afterimage, fusion on the synoptoscope, and visual acuity (fixation under 3 years of age). The patients were followed an average of 10 years. Treatment consisted of glasses and vision training. He found no correlation between the changes in refraction and the angle of squint. Of the patients with constant squint (116), only 6.7% had normal correspondence and 1% had functional cure. Of the patients with intermittent squint (26), 78% had normal correspondence and 85% had binocular fixation.

ESOTROPIA

Gittoes-Davies [21] described a new sydrome, with the following characteristics: unilateral esotropia of low degree (10 degrees ±); onset at about the age of 2½ years; onset after febrile disease, in 25% of cases; a family history, in 50%; no gross amblyopia, paresis, or vertical deviation; low hyperopia; central suppression; good fusion amplitude, and stereopsis. As possible etiologic factors,

17. Kretzschmar, S., Jr.: Measuring and Plotting of Ocular Deviations by Means of the Perimeter of Goldman, Ophthalmologica **123**:348-351, 1952.

18. Falkowska, Z.: Examination of Binocular Field of Movement on a Pane of Glass, Klin. oczna **21**:19-34, 1951; abstracted, Ophth. Lit. **5**:1187, 1952.

19. Thompson, D. A.: Measurements with the Cover Test Vs. Troposcope, Am. Orthop. J. **2**:47-52, 1952.

20. Nordlöw, W.: Spontaneous Changes in Refraction and Angle of Squint Together with the State of Retinal Correspondence and Visual Acuity in Concomitant Convergent Strabismus During the Years of Growth, Acta ophth. **29**:383-421, 1951.

21. Gittoes-Davies, R.: An Examination of the Actiology and Treatment of Small Convergent Deviations, Associated with a Low Degree of Hypermetropia, with a New Approach to the Treatment of This Condition, Brit. Orthop. J. **8**:71-84, 1951.

she suggests transient paresis of the ciliary muscle due to the toxin of febrile disease, with resultant spasm of convergence and an insufficient harmonic reflex. Prognosis would seem to be good because of the onset at an early age (2½ years) and the presence of all grades of binocular vision with good amplitudes; yet in none of the 80 cases did the usual treatment end in binocular single vision. She proposes the following treatment: If hyperopia is normal for the child's age, treat the patient as an emmetrope (she cautions against the use of glasses for more than two months if they fail to give binocular single vision, because the suppression is more intense if the glasses merely lessen the deviation); occlude for equal vision and to bring out the maximum angle; eliminate suppression at the maximum angle and elicit diplopia (the child is not to be taught relaxation or control); eliminate suppression on ductions and improve amplitudes; operate for the maximum angle; give postoperative orthoptic training along the usual lines.

In 1947, Franceschetti reported a case of acute concomitant strabismus in an adult. He now [22] adds five cases, of which the characteristics are as follows: Sudden onset with diplopia; constant angle; no paralysis or spasm of convergence; excellent fusion; insignificant refractive error, and probable preexisting exophoria. The cause is not known, but he speculates that it might be temporary impairment, by systemic disease, trauma, or stress, of fusion or divergence function, followed by an acute escape reaction, i. e., esotropia. He advocates prompt surgical intervention if orthoptic training is not successful in two months, and he obtains excellent results from small amounts of surgery. He emphasizes that diplopia does not always mean paralysis. The same syndrome is discussed by Meunier,[23] who reports another case. He postulates the same etiology except that he assumes a preexisting esophoria, an explanation which would seem more likely. Once decompensated, the large convergent angle represents an attempt to escape from the diplopia. He suggests anesthesia of the medial rectus muscles before resorting to operation but does not state how he effects it. He suggests as the name "concomitant strabismus with manifest diplopia."

EXOTROPIA

A symposium on intermittent exotropia, given at the joint meeting of the American Orthoptic Council and the American Association of Orthoptic Technicians in the fall of 1951, attempted to bring this subject up-to-date. Irvine,[24] in discussing the physiology, after mentioning many factors that do not explain it, concludes that convergence insufficiency is a lack of convergence tone and that divergence excess is a reversion to an atavistic type of innervation. Bair [25] gives a complete description of the condition and stresses the important part played by suppression for distance, making the binocular analysis unsatisfactory. She found that cases of exotropia made up 21% of strabismus cases and that of these cases of exotropia, 36% were of divergence excess and 24% were of combined divergence

22. Franceschetti, A.: Acute Concomitant Strabismus, Ophthalmologica **123**:219-226, 1952; abstracted, Am. J. Ophth. **35**:1379, 1952.

23. Meunier, M. A.: Acute Concomitant Strabismus, Tr. Soc. belge ophth. **101**:429-432, 1952.

24. Irvine, S. R.: Physiology of Intermittent Extropia, Am. Orthop. J. **2**:5-11, 1952.

25. Bair, D. R.: Diagnosis and Incidence of Intermittent Exotropia, Am. Orthop. J. **2**:12-17, 1952.

excess and convergence insufficiency. McPhail [26] describes the evolution of intermittent exotropia as divergence, then diplopia, then suppression. In the treatment this sequence must be reversed. She gives the orthoptic treatment in detail, emphasizing that suppression is often much deeper than is appreciated and that it must be removed before other treatment is attempted. Intensive, and often prolonged, orthoptic treatment is required. The best results are obtained by correlating orthoptic training and surgical treatment. Sugar [27] reviewed the entire treatment. He suggests that overcorrecting myopia may delay the development of convergence insufficiency. He has not found vertical prisms useful, but he advocates the wearing of dark glasses to relieve confusion and encourages near work. The only purpose of orthoptic training is to overcome, suppression. Surgery is the ultimate treatment and is usually required for exotropia of 20 △. He favors recession of one or both lateral rectus muscles. If there are normal findings for near, he does a small recession of one or both medial rectus muscles at the same time to prevent postoperative esotropia. In divergence excess combined with convergence insufficiency, in addition to antisuppression, orthoptic treatment aims at increased fusional amplitudes and improved convergence. In this condition he prefers resection and recession on one eye. If the deviation is over 50 △, he expects to operate on both eyes. Recognizing the difficulty of measuring the actual deviation, he does the cover test slowly and is careful to overcorrect with prisms.

ALTERNATING HYPERTROPIA

Schlossman,[28] in a study of 10 cases, found extreme variability of measurements. When combined with a large esotropia, the hypertropia may not become manifest until after operation. Surgery on the oblique muscles does not correct the hypertropia, and the author cautions against operations for the vertical muscles. Surgery on the horizontal muscles is less predictable than usual and should be undertaken only after prolonged study. He favors Posner's explanation that alternating hypertropia is an aberration of the postural tonus. Either fixation or change of one eye alone induces a change in the tonus equilibrium of both eyes.

Under the title "alternating hyperphoria," Crone [29] reports 80 cases with the following characteristics: upward deviation of the nonfixing eye; strabismus hyperphoria when looking to the side; rotation of both eyes around the visual axes in the direction of the covered eye, and rotary pendular nystagmus, which changes into a horizontal jerky nystagmus, with the rapid phase in the direction of the exposed eye when one eye is covered. He distinguishes three groups: (1) convergent strabismus with hyperphoria of the adducted eye; (2) convergent strabismus with hyperphoria of the abducted eye, and (3) divergent strabismus with hyperphoria of the abducted eye. Alternating hyperphoria associated with convergent strabismus and outward rotation of the covered eye is assumed to be due to failure of the optomotor reflexes originating from the lower nasal quadrants of

26. McPhail, A.: Orthoptic Treatment of Intermittent Exotropia, Am. Orthop. J. **2**:22-24, 1952.

27. Sugar, H. S.: Treatment of Intermittent Exotropia, Am. Orthop. J. **2**:18-21, 1952.

28. Schlossman, A.: Significance of Altering Hypertropia in Concomitant Strabismus, Am. J. Ophth. **35**:795-801, 1952.

29. Crone, R. A: Alternating Hyperphoria, Nederl. tijdschr. geneesk. **96**:54-56, 1952; abstracted, Excerpta med. XII **6**:396, 1952.

the retinas. Conjugated impulses aimed at compensation of this failure give rise to the abnormal position of the covered eye. Crone has had an unusually wide experience with these troublesome cases, so that his findings and conclusions are of considerable interest.

NONSURGICAL TREATMENT

Miotics.—To the 44 cases previously reported, Abraham [30] adds 88 cases in which miotics were used. Miotics were given only in cases in which the eyes had approximately equal refraction. Their use was of distinct advantage in 93% of the cases of periodic strabismus and in 35% of cases of constant strabismus. By "help from miotics," he means that the strabismus is absent or less frequent, with or without glasses. He believes that in most cases of convergent squint there is an accommodative factor. Miotics substitute peripheral accommodative action for both distance and near and give greater coverage than glasses. He has demonstrated a difference in amplitude of accommodation even with equal visual acuity. He uses miotics in treating amblyopic eyes, as he suspects weak accommodation in these eyes. Diisopropyl fluorophosphate is the best miotic, used in 0.01 to 0.03% concentration, and in his four years' experience he has seen no harm from use of this drug. Miotics may be used as a substitute for glasses in the very young, to help in making the change from glasses to no glasses, and to replace orthoptic training in cases of periodic strabismus.

Occlusion.—The investigative work on amblyopia has been reviewed earlier. For the treatment of amblyopia there seems to be complete agreement from all quarters as to the importance and success of occlusion. The trend appears to be toward starting it earlier and carrying it on longer (see "Results").

Baird [31] makes a very strong appeal for early treatment. He starts occlusion as early as the age of 4 months, in the belief that at that age the instability lends itself to corrective guidance. He occludes the fixing eye, thus breaking up any fixation pattern, improving rotations, and preventing suppression and abnormal retinal correspondence. Apparently, he continues occlusion until surgery is indicated, or until the child is old enough for orthoptic training. He implies that occlusion alone is successful in straightening the eyes in a significant number. He is convinced that the result of the treatment of nonaccommodative squint by occlusion and preoperative orthoptic training far surpasses surgery and glasses alone.

Carlevaro [32] advocates having a program for the detection and treatment of amblyopia in every nursery school. Such a program is already in operation in Milan.

A new method for treating eccentric fixation is offered by Fritz.[33] He believes that the image in the squinting eye usually falls on the blind spot, and he can demonstrate this by means of retinophotography. He occludes the fixing eye. The lens before the eye with eccentric fixation is treated as follows: The temporal half is

30. Abraham, S. V.: Use of Miotics in the Treatment of Nonparalytic Convergent Strabismus, Am. J. Ophth. **35**:1191-1195, 1952.

31. Baird, J. M.: Individual Correction of Strabismus, Am. Orthop. J. **2**:25-34, 1952.

32. Carlevaro, G. F.: Prevention and Treatment of Amblyopia ex Anopsia in Nursery Schools, Ann. ottal. e clin. ocul. **78**:147-155, 1952; abstracted, Am. J. Ophth. **35**:1895, 1952.

33. Fritz, A.: Treatment of False Projection in Concomitant Convergent Strabismus, Bull. et mém. Soc. franç. opht. **65**:381-387, 1952.

occluded; at the center there is a vertical 2 mm. clear strip covered with a red filter; a 7 mm. strip nasal to this is occluded; the remainder of the glass, nasally, is clear. The eccentric fixation is through the red strip, and images fall upon the macula through the clear portion nasally. He reports surprising results and cites the case of a patient, aged 22, whose vision improved from 1/20, with eccentric fixation, to 10/10 in four months.

Orthoptic Training.[34]—A discussion of the limitations of orthoptics by an orthoptist should be refreshing, but Wells [35] is a bit disappointing in this regard. She lists the following: (1) instrumentation, few limitations; (2) economic limitations, requiring that patients representing potential cures be selected for training; (3) psychological factors, such as cooperation and proper home influences, and (4) physiological factors, e. g., good general health. Amblyopia usually responds to occlusion; 3 months of treatment without improvement is her limit. She concludes that two factors are of great importance: (1) discontinuation of orthoptic training when there is no progress, and (2) careful selection of patients.

Portmann and Dufour [36] discuss the development of binocular vision and how strabismus interrupts this development. The disturbance is greater the earlier it occurs. The prognosis is worse the earlier the onset. They state that orthoptics has become an indispensable means of diagnosis and treatment in Switzerland. Their description of orthoptic treatment is in accord with the accepted views in the United States.

Rightly stressing the importance of psychology in orthoptic treatment, Feldman [37] gets the child's intelligence quotient, scholastic aptitude, and personality type from the school and then has an examination by a psychologist, so that the orthoptist can adjust the training to the child's intelligence. He believes that the orthoptist should be trained in education and psychology. No one will argue with this ideal program, but the cost and the time required, both in the training of the technicians and in the preparation of the patients, would seem to be prohibitive.

Under the title of "nystagmus treated by orthoptics," Healy [38] describes four patients with congenital nystagmus and strabismus. All the patients were hyperopic, and correction of any part of it accelerated the nystagmus. No abnormal retinal correspondence was found, probably owing to the constant movement. Occlusion exaggerated the nystagmus; yet vision improved more rapidly than usual. As the nystagmus disappeared when the subject was fusing at the angle of deviation, Healy believes that the nystagmus was an anomaly of the fusion mechanism.

34. Cronin, M. L.: Home Exercises, Am. Orthop. J. **2**:43-46, 1952. Gokhale, A. M.: Orthoptics, Indian Pract. **5**:264-266, 1952; abstracted, Ophth. Lit. **6**:355, 1952. Gorevan, S.: The Functions of the Orthopist, J. Irish M. A. **30**:72-74, 1952. Hill, J. C.: Modern Treatment of Squint in Children, Mod. Med. Canada **7**:27-29, 1952; abstracted, Ophth. Lit. **6**:177, 1952. Lee, B. M.: Some Recent Developments in the Treatment of Manifest and Latent Squint, with Particular Reference to the Use of Convergence Exercises, Brit. Orthop. J. **8**:87-94, 1951. Steckler, M. L.: The Eye Exercise Stick, Am. J. Ophth. **35**:1502-1503, 1952.

35. Wells, A.: Some Limitations of Orthoptics, Brit. Orthop. J. **8**:101-107, 1951.

36. Portmann, V. P., and Dufour, M.: Place of Orthoptics in the Treatment of Convergent Concomitant Strabismus, Ophthalmologica **123**:294-303, 1952.

37. Feldman, J. B.: Utilization of Psychology and Education in the Management of the Orthoptic Patient, Eye, Ear, Nose & Throat Month. **31**:89-95 and 103, 1952.

38. Healy, E.: Nystagmus Treated by Orthoptics, Am. Orthop. J. **2**:53-55, 1952.

Orthoptic training effected cures in these patients (one with operation), and afterward they could wear their refractive correction.

Sevrin and Callier [39] have obtained good results in the orthoptic treatment of exophoria. They describe their treatment in detail. In 80% of their cases the exophoria was corrected by orthoptics, which consisted chiefly of converging exercises on the synoptophore and home exercises, while in 8% surgical treatment was necessary in addition. The authors advance one or both medial rectus muscles if convergence is weak and recede the lateral rectus if there is too much divergence.

Two papers by orthoptists, in which they analyze their results, are of interest, although both series of cases are small. Scott [40] draws her conclusions from 51 surgical cases taken from an orthoptic clinic. She finds that the relation of age of onset to age at operation is of prognostic value. Patients with congenital squint rarely acquire binocular single vision if the operation is done after the age of 3 years. If the onset of squint is under the age of 2 years, there is a chance of binocular vision only if the operation is performed before the age of 3 years. When the onset is between 2 and 4 years of age, there is a 60% chance of binocular single vision. Preoperative training is valueless if the onset is before the age of 2 years. Postoperative training is indicated if there is some sign of fusion. If there has been abnormal retinal correspondence, one can rarely acquire strong fusion with depth. Scott feels that a large residual angle, even with a vertical squint, can be overcome by fusion, after operation. When the onset is before the age of 4 years, a preoperative difference in visual acuity of more than two lines precludes binocular single vision.

Engle [41] reviewed 40 orthoptic cases, 1 to 10 years after training. Patients discharged with abnormal retinal correspondence kept it. In 4 of 10 patients with a constant tropia on dismissal, the defect had become an intermittent tropia or phoria. Of 25 patients with intermittent tropias or phorias on dismissal, 4 had reverted to tropia; 21 still had good binocular single vision, and 4 of these showed improvement. She concludes that the majority receive benefit from orthoptic treatment and hold it.

Out of the many articles favorable to orthoptics, in one way or another, has come one discordant note. Douglas [42] has compared the results from two large orthoptic departments. He concludes that the greatest contribution made by orthoptics is to the knowledge and understanding of binocular reactions. There is no doubt about the usefulness of orthoptics in diagnosis, but he doubts whether the effort to use orthoptics therapeutically is worth while. In a second paper,[43] he surveys the results in 100 cases of concomitant convergent squint treated surgically and by orthoptic exercises. He doubts whether orthoptics, except for occlusion,

39. Sevrin and Callier: An Attempt to Treat the Disturbances of Convergence in Exophoria, Bull. Soc. belge ophth. **101**:432-439, 1952.

40. Scott, R. A.: Some Observations on the Expectation of the Development of Binocular Single Vision, Brit. Orthop. J. **8**:43-49, 1951.

41. Engle, D.: Strabismus Cases over a Period of Years, Am. Orthop. J. **2**:35-39, 1952.

42. Douglas, A. A.: Value of Orthoptics in the Treatment of Squint, Brit. J. Ophth. **36**:169-201, 1952.

43. Douglas, A. A.: Long-Term Results of the Treatment of Concomitant Convergent Strabismus in Terms of Binocular Function, Tr. Ophth. Soc. U. Kingdom **72**:383-393, 1952.

has any value in the treatment of squint. He believes that orthoptists should become "ophthalmic auxiliaries," whose function would be the detailed diagnosis of ocular function, to include perimetry and possible refraction. It should be added that from the two rather detailed papers it is apparent that Douglas is speaking from considerable experience with orthoptic clinics (See "Results").

Prisms.—The only mention of the use of prisms was that by Martyn,[44] who found that overaction of the superior oblique frequently accompanies convergence insufficiency and that orthoptic training will give only temporary improvement. Often the vertical deviation shows up only with convergence; so he uses vertical prisms, giving the amount found at the reading angle.

SURGICAL TREATMENT

Surprisingly few articles on the surgical aspect of strabismus appeared during the year. Sugar's[45] evaluation of his results in the use of measured recessions and resections is of interest because it gives the conclusions of this careful observer, drawn from 116 of his own cases. Contrary to some recent reports, he found that the greater the amount of total surgery done on a single eye at one time, the greater is the amount of correction per millimeter of surgical correction. In convergent strabismus, significant vertical deviations may cause marked overcorrections. Operation on one eye at a time permits reevaluation before the final procedure and necessitates the bandaging of only one eye. In spite of their marked variation, he feels that the surgical results follow a pattern that justifies working out a detailed guide for the amounts of resection and recession, in millimeters, for every 2 to 3 △ of deviation, from 10 to 45 △. Though he cautions that the values are not absolute, I question whether such figures have real value, even to one who is familiar with Sugar's technique.

Gibson[46] reiterates his confidence in the bilateral marginal myotomy. The patients that he considers favorable for this operation are (1) those with a reasonable probability of binocular vision, i. e., with age of onset after 1½ years, duration of squint of less than one and one-half years, age at operation less than 5 years, and similar findings (vision, fixation, excursions, and refractive error) in both eyes; (2) and those with a danger of exotropia after operation, i. e., very young patients, with onset under the age of 6 months, high hyperopia, and variable esotropia (accommodative, intermitten, and of recent origin). His objectives are (1) reduction of the excess action of the medial rectus muscles without impairment of normal function, (2) reduction of the deviation without postoperative exotropia, and (3) recovery of binocular vision. He gets the same effect regardless of the amount of the preoperative deviation. Of 43 cases, functional success was attained in 27%, cosmetic success in 62%, and an unsatisfactory outcome in 11%. These results would appear to be excellent.

44. Martyn, I.: Overaction of Superior Oblique Muscle, Tr. Ophth. Soc. Australia **11**:111-112, 1951.

45. Sugar, H. S.: An Evaluation of Results in the Use of Measured Recessions and Resections in the Correction of Horizontal Concomitant Strabismus, Am. J. Ophth. **35**:959-967, 1952.

46. Gibson, G. G.: Bilateral Marginal Myotomy, Tr. Am. Acad. Ophth. **56**:791-802, 1952.

Fischer [47] has given his choice of operation for convergent squint. The angle of squint and the range of abduction are measured on the perimeter. The patient's age is another important factor. Fischer divides his patients into three groups: 1. Those under 12 years of age with angles of deviation of less than 30 degrees, excess adduction and good abduction. Good results were obtained by tenotomy of the internal rectus. 2. Those over 12 years of age with medium to large angles of deviation and weakness of abduction. A combination of resection and tenotomy was used for this group. 3. An intermediate group with large angle of deviation and beginning weakness of abduction. Tenotomy of both medial rectus muscles gave good results. He worked out the following formula: $\frac{\text{Age} \times \text{Angle of Squint}}{\text{Abduction}} = X$ When X is small (less than 7), tenotomy is indicated. When X is large (more than 7), a resection-advancement procedure is indicated. When X is marginal (5 to 9) and the angle large, tenotomy of both medial rectus muscles, at two operations, is recommended; if the angle is small and abduction is poor, advancement of a lateral rectus is done.

Berens, Cole, Chamichian, and Enos [48] give a complete review of the inferior oblique. They point out that the theoretically large tortional variation that is introduced when its action is interfered with is not apparent practically. Less vertical correction is produced per millimeter of surgical correction of the inferior oblique than with a horizontal rotator because the major share of the difference introduced by surgery affects the tortional movements. They found the results of retroplacement more accurately judged and more permanent than those of tenotomy or myectomy. They give the following indications for retroplacement: (1) overaction of the inferior oblique on the same side as a paretic superior oblique in a nonfixing eye; (2) paresis of a superior rectus with the paretic eye fixing; (3) anatomical anomalies, (4) normal overaction of an inferior oblique, and (5) primary spasm of an inferior oblique. As a contraindication, they say that myectomy is preferable for hyperphoria or hypertropia over 10 D. unless surgery on the superior oblique is to be done. The technique of the operation is described. The results cannot be predicted. The authors obtained an average of 5 D. of correction in the primary position for 5.4 mm. of retroplacement.

It is interesting to find occasional references to some of the older operations that are not used much today in this country. Fischer's preference for a tenotomy of the medial rectus has been mentioned. Madroszkiewicz [49] has contrived a double mattress suture, made out of one long suture, which he uses as a safety measure with a complete tenotomy. Cavka [50] gives the results of a muscle-tucking operation in 27 cases of concomitant strabismus, 25 of which were of the convergent and 2 of the divergent type. If the angle is greater than 25 degrees, he does a tenotomy

47. Fischer, H.: Selection of Operative Method in Convergent Strabismus, Klin. Monatsbl. Augenh. **121**:153-160, 1952.

48. Berens, C.; Cole, H. G.; Chamichian, S., and Enos, M. V.: Retroplacement of the Inferior Oblique at Its Scleral Insertion, Am. J. Ophth. **35**:217-227, 1952.

49. Madroszkiewicz, M.: Muscle Recession with a New Safety Suture, Klin. oczna **21**:212-214, 1951; abstracted, Ophth. Lit. **5**:1190, 1952.

50. Cavka, V.: A Muscle Tucking Operation in Cases with Concomitant Squint, Med. Arch. **2**:5-11, 1951; abstracted, Ophth. Lit. **5**:1030, 1952.

of the antagonist as well. A complete correction was achieved in all but three cases. He considers the tucking procedure surer than advancement with resection.

Borsello [51] has studied the variations of the corneal curvature following an advancement. This operation on the medial or the lateral rectus was usually followed by an increase in the refraction of the horizontal meridian of the cornea and by a decrease in the vertical meridian. He followed 20 patients an average of 58 days. I question whether this is a sufficient interval after operation, for it has been my impression that this change is only temporary (See "Exotropia").

RESULTS

The long-term results of the treatment of concomitant convergent strabismus in terms of binocular function was the subject of a symposium at the Oxford Ophthalmological Congress in July, 1952. Cashell [52] opened the symposium. He had gathered cases from 18 hospitals in all parts of the United Kingdom. The patients had been discharged in 1949. A detailed questionnaire is presented. His criteria for a satisfactory binocular result are that the patient should have binocular vision for distance and near, with glasses if necessary; that if the cover test shows a phoria, immediate recovery must occur on removal of the cover, and that the binocular vision should be one of fusion and stereopsis. No cases of intermittent strabismus were included. There were 1,119 patients, and 33% had been discharged with binocular vision. Of 820 operations, the combined recession and advancement procedure was the most popular. There was no relation between the amount done and the amount of correction. The obstacles to binocular vision that he found were, chiefly, inadequate surgery, excessive surgery, postoperative vertical deviation, persistent amblyopia, and abnormal retinal correspondence. He concludes that the sooner the visual axes are restored to parallel, by all means, the better the prognosis for binocular vision. This survey must have been a staggering piece of work. To attempt to analyze such a large number of cases from so many different sources is a task almost beyond comprehension. It is regrettable, but understandable, that more valuable conclusions could not have been drawn from it.

Douglas [53] continued the symposium, reporting on 100 of his own patients treated by orthoptic exercises and operation. Thirty-six had been discharged as cured, but he expresses doubt whether some of the earlier ones were really cured. Twenty-two of these still had straight eyes, but only 2 had truly normal binocular function. Of 64 for whom the results were only cosmetically satisfactory on discharge, 13 had acquired binocular single vision. He concludes that the age of onset is the most important factor in determining good physiological results, the later the better. Patients discharged with good binocular function had maintained it; those without it were not handicapped in any obvious way. As mentioned earlier, he doubts whether orthoptics has any value in the treatment of squint.

In the discussion, Whittington [53] observes that if one takes care of the function, the appearance will take care of itself. Results depend on the age of onset. He

51. Borsello, G.: Variations of the Corneal Curvature Following the Advancement of Internal or External Rectus Muscles, Rass. ital. ottal. **21**:55-62, 1952.

52. Cashell, G. T. W.: Long-Term Results of the Treatment of Concomitant Convergent Strabismus in Terms of Binocular Function, Tr. Ophth. Soc. Kingdom **72**:367-381, 1952.

53. In discussion in Symposium on Long-Term Results of Treatment of Connecticut Convergent Strabismus in Terms of Binocular Function, Tr. Ophth. Soc. U. Kingdom **72**:395-409 1952.

favors early operation, but in the case of a patient with alternating squint he will defer operation for fusion training. Houlton [53] comments on 104 cases of alternating squint from Cashell's survey. Twenty-seven patients were discharged with binocular vision. He found that a large proportion of true alternators had the onset at birth or soon after. Those who do acquire binocular vision probably had some development of binocular reflexes before the onset of the squint. He thinks that orthoptic training makes very little difference in alternating squint, especially when it is of early onset.

In the same symposium, Lyle [53] reports 172 of his private cases. Restoration of normal binocular function was achieved in 44%, in about one-third of which no surgical procedure was carried out. He finds two questions of prognostic value: At what age did the patient exhibit his squint? What is the patient's age now? In the accommodative group, if the eyes are straight for distance but there is an esotropia for near, operation, he believes, is usually indicated after the age of 4 years. In the nonaccommodative group, he favors operation as soon as the patient is made a free alternator. He considers preoperative orthoptic training of doubtful value, but after operation it may be invaluable. If the eyes are straight within one year of the onset, the prognosis for binocular vision is fairly good. He believes that there is probably little to be gained by operating before the age of 15 months.

Anderson,[54] reporting from Australia, analyzes over 300 cases of convergent strabismus. This is a detailed report, and only his more striking conclusions can be given. Emphasizing that amblyopia is much too common, he found that no patient with a difference of two lines in the vision of the two eyes was cured. He believes that occlusion is the most important and the most neglected treatment of squint. Abnormal retinal correspondence can be cured at a much older age than amblyopia. Twelve training periods before operation are sufficient if normal correspondence is to be obtained. If the amount of orthoptic treatment that can be given must be limited, it should be saved until after the operation. After the history taking, refraction, and occlusion, he operates if the squint is constant and not accommodative. In gross cases he will operate during the second year, and will operate a second time for a vertical deviation of over 5 △. He continues occlusion until orthoptic treatment is well advanced. He states that cures should be established by the fifth year, indicating that apparently he is able to use orthoptic training at an earlier age than many of us (See "Orthoptics").

54. Anderson, J. R.: A Cross-Section of Cross-Eyes, Brit. Orthop. J. **8**:14-33, 1951.

Society Transactions

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

James W. Smith, M.D., Presiding in Absence of Willis S. Knighton, Chairman

Raymond E. Meek, M.D., Secretary

Combined Meeting with Clinical Society of the New York Diabetes Association

Nov. 7, 1952

Diabetes Retinitis: The Internist's View. Dr. Joseph T. Beardwood Jr., Philadelphia (by invitation).

Type of Diabetes Mellitus Associated with Diabetic Retinitis. Dr. George W. Dana, Baltimore (by invitation).

A study was made of 190 cases of diabetes mellitus in which autopsies were done in recent years at The Johns Hopkins Hospital. In 57 of these the Kimmelstiel-Wilson lesion was present. It was concluded that adult patients who have the Kimmelstiel-Wilson renal lesion (intercapillary glomerulosclerosis) also have diabetic retinitis of the capillary-aneurysmal type. Their diabetes often tends to ameliorate and is characterized by rarity of acidosis. The question then arises whether the metabolism of such diabetics differs from that of ordinary diabetics.

Since diabetic patients with complete lack of insulin rarely exhibit the Kimmelstiel-Wilson syndrome and since, on the other hand, diabetic patients with, presumably, a near-normal supply of endogenous insulin do show the specific capillary and renal damage to an extensive degree, it must follow that insulin deficiency alone does not explain the capillary defect. Could it be due to a factor that is concerned with obesity or hyperlipemia or to an excess of a hyperglycemia-producing substance?

Four hormones when present in excess are known to cause hyperglycemia. One is the growth hormone from the anterior lobe of the pituitary; a clinical instance of the effect of an excess of this substance in the adult is acromegaly. Epinephrine is another, deriving from the adrenal medulla; this in excess may produce pheochromocytoma. These hormones in pathological increase may cause diabetes, but their clinical effects have not been characterized by diabetic retinitis. Adrenocorticosteroids in higher than normal amounts often cause hyperglycemia, as in Cushing's syndrome; yet a patient with such a condition rarely shows diabetic retinitis. However, the elective use of corticotropin or cortisone in the treatment of diabetic retinitis is not well regarded. The fourth hormone to be considered is the hyperglycemia factor, deriving supposedly from the alpha cells of the pancreatic islets. This potent hormone produces hyperglycemia by accelerating hepatic glycogenolysis.

One assumes that in the normal person the pancreatic α-β-cell output is in homeostasis. Beta-cell depletion with underproduction of insulin, if not corrected by an exogenous supply of insulin, leads to diabetic acidosis and coma. Traditionally, this is thought of as the basis of diabetes mellitus. However, what might be the effect of the hypothetical converse, in which insulin production was not impaired but a true excess of the hyperglycemia factor developed? In such a diabetes the endogenous insulin supply would be expected to be adequate and acidosis would be avoided, but the supply of insulin would be insufficient to control the abnormal hyperglycemia, especially if the hyperglycemia factor were correlated with an insulin-antagonistic effect. This condition is hypothetical, and the data for its acceptance or denial are still unobtained. Thus far, no clinical entity

associated with demonstrated excess output of hyperglycemia factor has been delineated, and the relationship that may exist between adrenocorticosteroids and the hyperglycemic factor in the production of capillary damage remains to be seen. But it is difficult to account for the hyperglycemia characterizing patients with capillary aneurysms solely on the basis of insulin deficiency. Furthermore, there are occasional cases of advanced retinitis of diabetic type in patients with normal blood sugar levels and normal glucose tolerance.

In conclusion, this capillary disease of the retina at its onset appears without arteriolosclerosis. Presumably, it shares its pathogenesis with the Kimmelstiel-Wilson intercapillary glomerulosclerosis. Its highest incidence occurs in diabetes mellitus with rarity of acidosis, the hyperglycemia of which seems not to be explained by insulin deficiency and the progress of which seems undeterred by insulin administration. It is likely that the pathogenesis of the capillary damage derives from a disturbance of carbohydrate and lipid metabolism other than a deficiency of insulin.

Diabetic Retinitis: The Ophthalmologist's View. Dr. Jonas S. Friedenwald, Baltimore.

Diabetic retinopathy is characterized by the presence in the retina of great numbers of saccular capillary aneurysms, some of which have hyalinized walls and closely resemble the hyaline nodules in the glomerulus described by Kimmelstiel and Wilson. The retinal and renal lesions commonly occur together, are manifestations of the same disease process, and are not dependent on atherosclerosis, hypertension, or arteriolosclerosis. The etiology of this vascular lesion is still obscure. Rich and associates have produced lesions resembling Kimmelstiel nodules in the kidneys of rabbits treated with cortisone. Alloxan-diabetic rabbits which I have treated with cortisone or corticotropin exhibit retinal capillary aneurysms and hyaline glomerular nodules. The vascular lesion of the diabetic may be due to an interaction between the metabolic abnormality of diabetes and adrenocortical function.

DISCUSSION

Of Papers by Drs. Beardwood, Dana, and Friedenwald

Dr. Howard Frank Root, Boston: Those interested in diabetes and the detection of diabetes are more concerned about retinitis than any of the other complications or sequelae of the disease. In Boston we find that the number of diabetics on the Registry of the Blind, on Tremont Street, increased eight times between 1945 and 1951. In our survey of our young people, we found that for every one who was legally blind there were 70, not 7, with retinitis in one of various stages of development.

I am tremendously interested in what Dr. Beardwood said about the relation of treatment to the disease; I should like to say a word about the natural history of diabetes. He has made the case clear. Ashton, I think, is the first ophthalmologist who, on the basis of his own analysis of the character of treatment, arrived at the same conclusion regarding the control of diabetes. What is the natural history of retinitis proliferans? Our series as of last week had reached a total of 458 patients with retinitis proliferans. What is the history of these patients? The vast majority of them had the beginning of their diabetes in childhood or in early life. Retinitis proliferans in a diabetic whose disease begins after the age of 50 is practically unknown. Retinitis proliferans is a disease of youth. Diabetes in youth is severe. The first 5 or 10 years of the disease does not differ from the diabetes of any youthful person. After 6 to 10 years of diabetes, when the patient gets out from under his mother's apron strings, he is no longer controlled, and then acidosis comes into the situation. He breaks treatment and subjects himself to a variety of stresses and strains. Among the conditions which may develop is pyelonephritis, which Dr. Dana showed accompanies the Kimmelstiel-Wilson lesion. I, personally, should like to minimize the Kimmelstiel-Wilson lesion, because it concentrates

attention on a lesion which is probably the least important of those comprising diabetic nephropathy. In these patients with retinopathy it is lack of control which precedes the retinal involvement. From observations on these 458 patients, we obtained the following facts: Of 188 patients whose diabetes began early in life and has lasted 20 years, so that they are now 25 to 38 years old, an examination of the eyegrounds showed that in 15%, who have had good control, there was no retinitis and no nephropathy, but that in the group with poor control, as Dr. Beardwood described, nephropathy was present in 25% and retinitis in 65 to 70%. Bornstein pointed out that the lack of insulin in the blood, demonstrated by his method, and in the pancreas, as shown at autopsy, is found not in childhood alone but at any age, provided the patient, old or young, has had uncontrolled diabetes, that is, acidosis and great loss of weight. He did not say that diabetic patients commonly had normal blood insulin; rather he stated that in certain middle-aged persons with mild diabetes who had not had ketosis he found 70% of the normal amount of insulin in the blood. I beg the ophthalmologists to get as accurate records as possible on their patients: When did the diabetes really begin? What kind of diabetic treatment did the patient have? Within the last six months we have had the following experience: One of our patients, aged 28, who had had diabetes for 18 years came in with typical diabetic nephropathy (Kimmelstiel-Wilson lesion), edema, albuminuria, and retinitis proliferans. We did the best we could for him, which was not much. In 10 days he returned to the New England Deaconess Hospital in diabetic coma, with profound ketosis, having lost 20 lb. (9.1 kg.) of edema fluid. In 72 hours after he recovered from the ketosis his edema returned, and he died in uremia within 30 days.

One of the reasons not sufficiently stressed that a patient after diabetic nephropathy has a lowered insulin requirement is his improved dietary practice. There is no weapon so effective in promoting dietary control as the loss of an eye or the announcement that retinitis has made its appearance.

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OPHTHALMOLOGY

George F. J. Kelly, M.D., *Chairman*

M. Luther Kauffman, M.D., *Clerk*

Jan. 15, 1953

Open-Angle Glaucoma. Dr. W. Morton Grant (guest speaker) and Dr. Robert R. Trotter.

In this report the authors presented their current views and correlated the results of their own investigations with those of others. The term "open-angle glaucoma" is preferred to the older "chronic simple glaucoma" as being more specific and descriptive. The preferred name emphasizes a fundamental distinction from "angle-closure glaucoma," on which there is good agreement among the conclusions of Scheie, Haas, Chandler, and the present authors.

In open-angle glaucoma, the angle of the anterior chamber remains open at all times, although the width of the angle varies in different patients from wide to narrow.

As in other glaucomas, there is in open-angle glaucoma an abnormally great resistance to the outflow of aqueous humor. In open-angle glaucoma the resistance characteristically remains abnormal during spontaneous fluctuations of intraocular pressure, even into the normal-pressure range. This contrasts with the situation found in angle-closure glaucoma, in which abnormal resistance is present only during episodic attacks of obstruction of the trabecular drain by the iris or during chronic closure by peripheral anterior synechia.

An abnormality of resistance to outflow has long been recognized as a characteristic of glaucoma and has formed the basis for several diagnostic tests, such as

the water-drinking and compression tests. A test employed by Kronfeld, utilizing compression and pressure measurement with a Schiøtz tonometer, was one of the forerunners of a test by Moses and Bruno and of a current method ("tonography") devised by the authors. In the authors' test, an electronic tonometer and recording galvanometer are employed to measure the fall of pressure which occurs while the tonometer is allowed to rest on the patient's cornea.

A graph in which the initial tonometer-scale reading is plotted against the reading after four minutes of tonometer application for a large group of normal eyes and for a group of definitely glaucomatous eyes shows that the two groups fall in separate areas of the plot, with only a moderate overlap. It appears that on this basis eyes with open-angle glaucoma can be fairly well distinguished from normal eyes, even at a time when the pressure itself is within the normal range. (These findings do not, of course, apply in angle-closure glaucoma, glaucoma secondary to inflammation, or in glaucomatocyclic crisis at times when the pressure is normal.)

This method of measurement has clinical usefulness in the detection and diagnosis of glaucoma when employed as a qualitative test, like that of Kronfeld. However, only through mathematical analysis of the measurements does it provide an answer to whether the abnormal resistance in glaucoma is a minor, inconsequential characteristic of the disease or whether it is the major abnormality. Such an analysis, based on Friedenwald's data for pressure and volume relationship and on the authors' new and independent data, indicates that abnormal resistance to outflow, rather than an abnormality of rate of aqueous formation, is the principal reason for the elevation of intraocular pressure.

The cause of obstruction to outflow is apparent in angle-closure glaucoma, as well as in the open-angle glaucomas with abnormal material visible in the trabeculum, as in glaucoma capsulare and in pigmentary glaucoma. However, in primary open-angle glaucoma in which no abnormality is visible in the angle, it seems that the impediment to outflow must be explained by abnormality of the aqueous-outflow channels themselves or by elevated venous back-pressure in the recipient vessels. The evidence is principally in favor of an abnormality of the outflow channels. The same kind of tonographic finding of resistance is obtained in primary open-angle glaucoma as in the glaucomas due to obvious mechanical obstruction. In some of these other glaucomas the resistance is found to persist after enucleation, with the venous back-pressure zero. In the case of intraocular pressure elevated in association with increased venous pressure, such as is found in patients with arteriovenous fistula, the tonographic measurements differ from those of primary open-angle glaucoma and do not indicate abnormal resistance to outflow. Increase in venous back-pressure and increase in intraocular pressure are essentially equal in amount. This parallelism has been shown by Weekers in a case of arteriovenous fistula and is also true in measurements on enucleated normal eyes when the back-pressure has been varied experimentally. In glaucomatous eyes with elevated intraocular pressure no comparable rise in venous pressure has generally been found by those who have made measurements on recipient vessels in patients.

The authors conclude that abnormality of the outflow channels themselves is responsible for the increased resistance to outflow in glaucoma, and it is the nature of this abnormality which is now being most actively investigated.

The site of abnormality has not been determined. However, there is evidence that very little of the resistance to outflow in normal eyes is associated with structures in the conjunctiva, episclera, or outer half of the sclera, for these structures may be dissected away without altering the outflow resistance in enucleated normal human eyes. The outflow resistance in these eyes is essentially the same as that found by tonography in normal eyes of patients.

Treatment is now principally directed toward improving the facility of aqueous outflow, either by drug or by surgical means. It is possible that as a better understanding of the cause of obstruction to outflow is developed, more effective measures may become apparent.

News and Comment

EDITED BY DR. W. L. BENEDICT

GENERAL NEWS

Annual Clinical Conference, Wills Eye Hospital.—The Sixth Annual Clinical Conference of the Wills Eye Hospital will be held on Feb. 19 and 20, 1954. Color television, sponsored by Smith, Kline & French Laboratories, will again be a feature of the program.

Retina Service Fellowship.—This fellowship has been created by the Retina Foundation and will be granted to a young ophthalmologist from the United States, or abroad, who wishes to be trained for 6 to 12 months at the Retina Service of the Massachusetts Eye and Ear Infirmary. The remuneration will amount to $1,000 per annum, plus room, board, and laundry at the Infirmary.

Training covers methods of examination of the extreme periphery of the fundus and the vitreous; use of the indirect stereoscopic opthalmoscope and scleral depressor, and surgical procedures in cases of retinal detachment. Original research of a clinical nature will be encouraged. The only candidates who are eligible are those who have completed a residency in ophthalmology or have had comparable training.

Applications, accompanied with a biography and at least two references, should be mailed to Dr. E. B. Dunphy, Chief of Ophthalmology, Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston 14.

SOCIETY NEWS

Colorado Ophthalmological Society.—The summer convention of the Colorado Ophthalmological Society will be held on Aug. 3, 4, 5, and 6, 1953, in conjunction with the Department of Graduate Education of the University of Colorado School of Medicine. Guest speakers, seminars, and demonstrations will constitute the four-day meeting, to be held at the University of Colorado medical center, in Denver. Membership in the Society and attendance at the meeting are open to all interested and accredited physicians. For information on registration and fees, write to the Director of Graduate Education, University of Colorado School of Medicine, 4200 E. 9th Ave., Denver.

PERSONAL NEWS

Appointment of Dr. William M. Hart to the Public Health Service.— Dr. William M. Hart, assistant professor of Ophthalmology, Jefferson Medical College of Philadelphia, has been appointed Chief of the Ophthalmology Clinics of the National Institute of Neurological Diseases and Blindness, Public Health Service. His appointment began as of April 15.

Books

Transactions of the American Ophthalmological Society. Volume 50, 1952. Price, $18. Pp. 740. Columbia University Press, 2960 Broadway, New York 27, 1953.

The fiftieth volume of the *Transactions of the American Ophthalmological Society* reports the proceedings of the Society and the papers presented at the Eighty-Eighth Annual Meeting, held at Hot Springs, Va., in 1952.

The following papers were presented:

Daviel, Modern Surgeon, H. Rommel Hildreth, M.D.

Preanesthetic Sedation and Analgesia for Intraocular Operations Done with Local Anesthesia, Walter S. Atkinson, M.D.

The Surgical Treatment of Glaucoma Complicating Congenital Aniridia, Eugene M. Blake, M.D.

Corneal Contact Lenses, Conrad Berens, M.D., and (by invitation) Louis J. Girard, M.D., and Lieut. (jg.) Kenneth Foree (MC), U.S.N.R.

Carcinoma of the Orbit and Adnexa, William L. Benedict, M.D.

Congenital Paralysis of Medial Rectus Muscle with Unusual Synergism of the Horizontal Muscles, Hermann M. Burian, M.D., and (by invitation) John E. Cahill, M.D.

Pigmentary Glaucoma and Its Relation to Krukenberg's Spindles, F. Phinizy Calhoun Jr., M.D.

Etiologic Considerations of Vertical Muscle Defects, Walter H. Fink, M.D.

Congenital Stationary Night Blindness Without Ophthalmoscopic or Other Abnormalities, Frank D. Carroll, M.D., and (by invitation) Charles Haig, Ph.D.

Indications and Contraindications for Adrenal Cortical Hormone Therapy in Ocular Disease, Alan C. Woods, M.D.

Angiomatosis Retinae (von Hippel's Disease) Eleven Years After Irradiation, Frederick C. Cordes, M.D., and (by invitation) Ariah Schwartz, M.D.

Glaucoma with Cataract Surgically Cured by Single Operation, Henry L. Birge, M.D.

Atopic Keratoconjunctivitis, Michael J. Hogan, M.D.

Spontaneous Absorption of Cataract, Searle B. Marlow, M.D.

Prevention of Infection in Ophthalmic Surgery: Further Studies, M. Hayward Post Jr., M.D.

A Remarkable Case of Bilateral Neuroretinal Edema, Arthur J. Bedell, M.D.

Detachment of the Vitreous as Seen with Slit-Lamp Examination, Dohrmann K. Pischel, M.D.

The Ascorbic Acid Content of the Aqueous of Surgically Aphakic Human Eyes, Peter C. Kronfeld, M.D.

Surgical Correction of Spastic Senile Entropion: A New Method, Daniel B. Kirby, M.D.

Intraocular Biopsy: An Evaluation, T. E. Sanders, M.D.

Retinal Changes Associated with Coarctation of the Aorta, Glenn L. Walker, M.D., and (by invitation) Thomas F. Stanfield, M.D.

Five theses were accepted as part of the requirement for candidacy in the Society. These are as follows:

The Role of the Sex Chromosome in Hereditary Ocular Pathology, Harold F. Falls, M.D.

Beta Radiation Therapy in Ophthalmoscopy, William F. Hughes Jr., M.D.

Stability of Aqueous Solutions Commonly Employed in the Treatment of Primary Glaucoma, W. H. Morrison, M.D.

Alpha Irradiation, Robert N. Shaffer, M.D.

Fundoscopic Localization, George H. Stine, M.D.

The Purpose of the Guild

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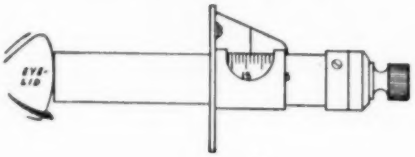

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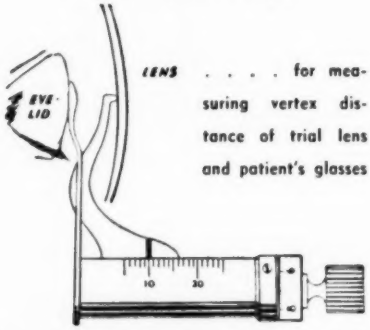

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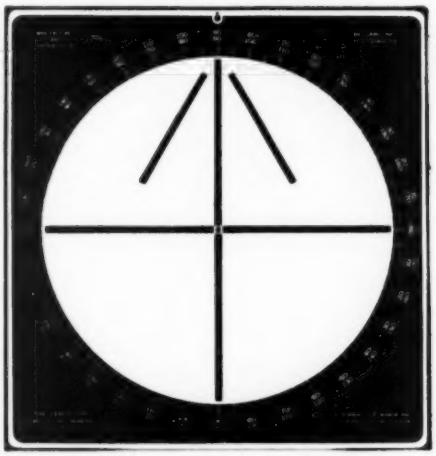

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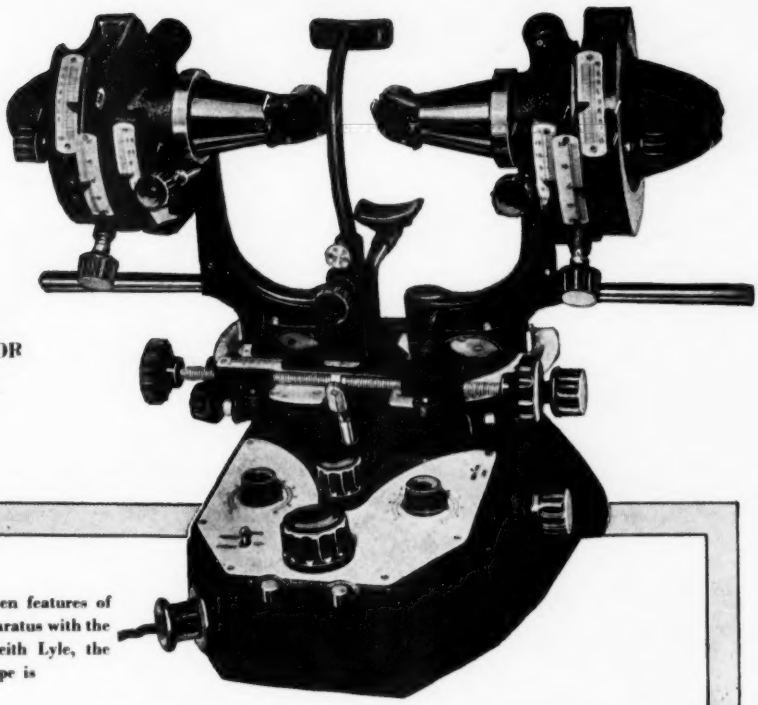

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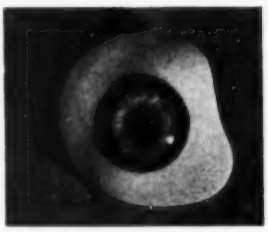

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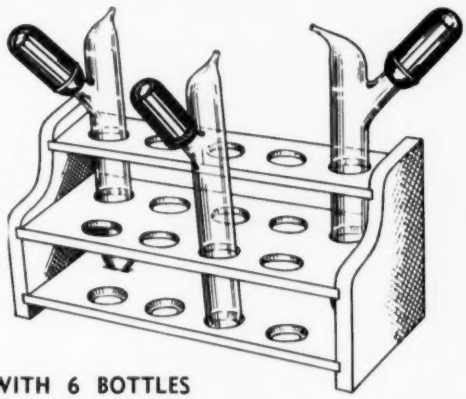

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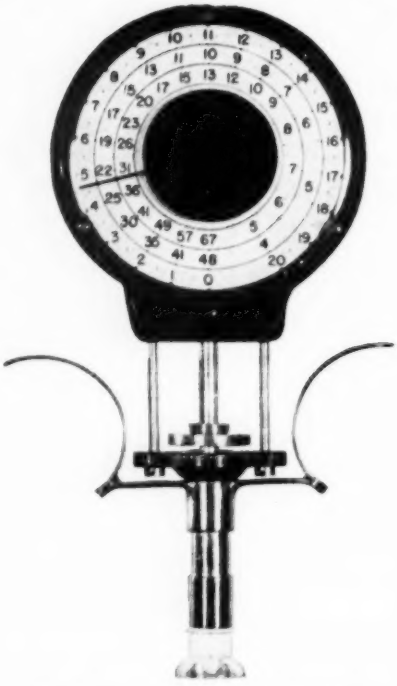

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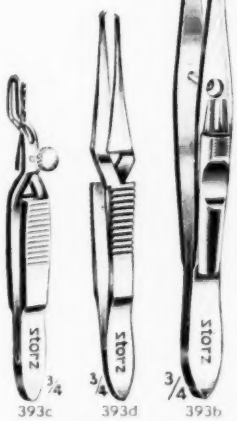

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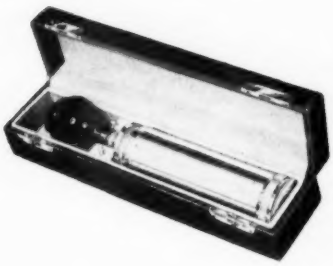

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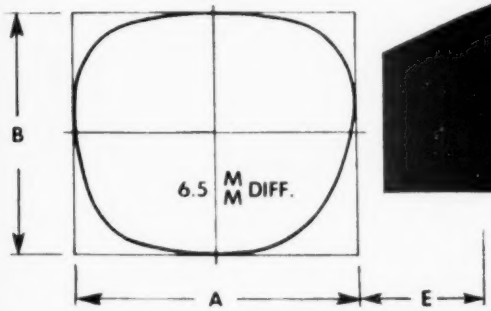

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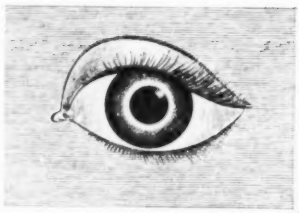

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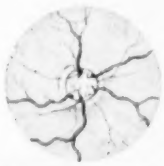

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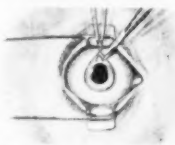

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